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The New York Academy of Medicine.

By

The Society of the New York Hospital,

March, 1898.

This is a case of glaucoma, with the usual well-marked ophthalmoscopic appearances. What is most remarkable is the peculiar and rare emerald-green hue of the disk. (See text, pages 218 and 232.)

TEXT-BOOK

OF

OPHTHALMOSCOPY.

BY

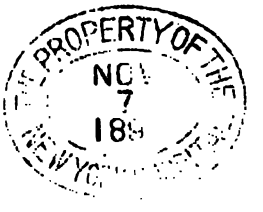
EDWARD G. LORING, M. D.

EDITED BY

FRANCIS B. LORING, M. D.

PART II.

DISEASES OF THE RETINA, OPTIC NERVE, AND CHOROID,
THEIR VARIETIES AND COMPLICATIONS.



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EDITOR'S PREFACE.

Dr. E. G. Loring, the author of this work, died suddenly on the 23d of April, 1888, before its completion. It was my original intention, on assuming the charge of the manuscript that he left, to complete it, simply in order to present it in a finished state to the public. After a careful reading of it, however, I found that there was so much original matter in it, so much that from its very nature must provoke discussion and argument, that I determined to publish it as it stood, without addition or correction. This determination was further strengthened by my subsequently finding a note-book, the contents of which, beginning at the section on Glaucomatous Excavation, carry the book practically almost to its close. This latter portion has been added exactly as it was taken from the penciled notes; and if the reader misses the elaboration and finish that have characterized the previous writings of the author, he must remember that it has not had his final supervision, and that much that might have been done for the sake of elegance has been left undone, lest it should interfere with the clearness of his expression of professional matters, or misinterpret his statements in regard to facts.

So, too, with the illustrations: it was Dr. Loring's habit to draw these for himself; and though, while most of those called for by the text were finished, some were not, and their places have had to be filled with those from other authorities. The large collection of ophthalmoscopic cases that were to have been added as plates were, many of them, unfortunately not drawn; and, as no record of them has been kept, it has been impossible to reproduce them.

In conclusion, I desire to acknowledge the obligation, in preparing this work for the press, that I am under to Dr. O. F. Wadsworth, of Boston, and Dr. R. H. Derby, of New York, whose lifelong friendship for the author has been so fully expressed by their kindness to the editor.

F. B. L.

Washington, D. C., *February, 1891.*

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TEXT-BOOK OF OPHTHALMOSCOPY.

CHAPTER I.

THE RETINA.

Disturbances in the Vascular System.—When pressure is exerted upon the outside of the ball of the normal eye, especially upon one in which previous to such pressure there is no visible movement in the circulation, the equilibrium which existed between the pressure upon the outside of the vessel and that within is disturbed, and as a result many interesting phenomena are observed; and it will be noticed that these manifestations are in direct proportion to the amount of pressure exerted, and that a return to the original state is also proportionate to the rapidity with which the pressure is relaxed.

When the minimum amount of pressure of the finger is exerted which is capable of producing a noticeable effect, it will be seen that this effect is shown first not upon the arteries but upon the veins, and that the pulsation which follows is rhythmical with the action of the heart; and, what it is most important to observe, that the pulsation takes place not only first but most forcibly upon the surface of the disk, and is most apparent at the porus opticus. When the force is increased, it will be observed that, although the veins empty themselves under the pressure from the centre toward the periphery, and thus become smaller and less visible upon and near the disk, nevertheless, the absolute amount of pulsation is not increased *pari passu* with the pressure, but, on the contrary, it is lessened. When the pressure is carried to the fullest extent consistent with the safety of the organ, that portion of the vein which lies nearest to the disk is completely emptied of its contents, and all pulsation ceases, as the vessel is entirely collapsed. This pulsation does not cease abruptly, but gradually, from the centre toward the periphery, and can be followed just before it is lost to view for a little way over the surface of the retina.

Turning now to the arteries, we see that similar effects also take place, though the character of the pulsations is somewhat different. It is much more rapid and much more vivid to the eye, appearing in some cases almost to flash, and the effect of the pulsation can be traced farther into the retina. It is manifested in the neighborhood of the disk by the ordinary collapsing and refilling of the vessel, and at a greater distance from the nerve by a slight but still perceptible excursion of the vessel.

In the mean while important changes have taken place in the calibre both of the veins and the arteries. Very little, if any, change seems to occur under light degrees of pressure, as, for example, such as are just sufficient to call forth a perceptible pulsation, though the antero-posterior curve of the vein as it drops down into the porus opticus is seen to be changed, especially where this curve is pronounced. This is shown by the fact that the light-streak, or, as it is commonly known now, the reflex from the summit of the vessel, has disappeared. This is due in part to the change in curvature, through which the rays of light from this portion of the vessel do not return to the observer; and partly, I think, from changes in the fluid itself which cause the blood at being retarded just at this point to assume a darker color. If the pressure is now increased a little, so that the current becomes retarded to a greater degree, the form of the vessel is seen to undergo a change. The veins become fuller, less flat, or, in other words, more cylindrical in shape. If the pressure is augmented still further, the veins begin to show a tapering condition toward the disk, and it will be seen on careful examination that they are fuller and proportionately larger at the periphery of the field than at the disk; and that, even if the pressure is increased to its fullest extent, the veins can not be emptied at the periphery of the field, or even perceptibly reduced in size, long after they have collapsed entirely and become invisible upon the disk and its immediate neighborhood. It must be observed here that it requires no little skill to keep the vessels under observation while the higher degrees of pressure are exerted upon the eye, as not only is the patient apt to move the eye, but also the relations of the details of the fundus are much changed and obscured.

It will be seen from this that a pressure on the outside of the walls of the veins sufficient to overcome the resistance of the column of blood within, while it reduces the quantity of blood near the centre of the field, has a tendency to maintain or even increase it at the periphery; or, in other words, the blood is backed up toward the capillaries, because its exit from the eye is impeded or stopped.

Turning again to the arteries, we see that the effect of the pressure is manifested in an entirely different way in some respects. Instead of the arteries becoming fuller and more cylindrical under a moderate pressure they become smaller and flatter, and on an increase of pressure they become suddenly and abruptly invisible upon the disk, though they still remain visible at the periphery of the field, as if in shutting off the supply at the disk the blood which had already entered the vessel had been driven onward toward the capillaries just as the venous blood had been driven backward toward them.

It will have been noticed by the careful observer that in poportion as the pressure is increased, the light-streak gets smaller and finally disappears. This is due probably to two facts: first, because the vessel becomes fuller and more cylindrical, and the curve therefore sharper; and, second, because when the current is retarded or entirely stopped, not only is the transparency of the wall interfered with, but also the contents of the vessel in their relations and proportions are changed, so that so much light is absorbed that sufficient does not return to the observer to produce the light-streak. Or it may be that the impediment to the circulation changes also the nature and consistency of the fluid in the lymph-spaces which surround the vessel, or that the relation between the plasmic current and the red current is changed so that the fluid becomes more mixed and less transparent. Be that as it may, it is certain that the disappearance of the light-streak upon the veins is due to an altered condition of the blood in the *veins*, as is shown by the fact that the same thing does not occur in the case of the arteries. On the contrary, the light-streak becomes broader and broader under pressure, in proportion to the diameter of the vessel, until the vessel entirely disappears. The moment the pressure is relaxed and the current re-established, the light-streak returns to the veins. It will be seen, too, that under the effect of pressure the color of the blood has changed; that as the current becomes slower, the current gets darker in the veins and lighter in the arteries. The darker color in the veins is probably due to the fact that, as the current is slower, the blood remains longer in contact with the walls of the vessel; its oxygen is consequently more exhausted in the greater metamorphosis of tissue, and the accumulation of detritus is greater.

The lighter color in the arteries is due mostly to the reduction in size of the blood-column, and especially in the reduction of the antero-posterior or perpendicular diameter of the vessel. As the blood is prevented from entering the eye by the pressure, the vessel becomes flatter, more ribbon-like, and the color is consequently lighter and

the reflex broader. This takes place to a greater degree near the disk, because the pressure from anatomical conditions produces its greatest effect at this place; so that at the very time that the larger arteries at the disk have collapsed so that no blood enters them, the smaller vessels near the periphery of the field still maintain a considerable if not normal degree of fullness, and the color of the blood is therefore somewhat darker. Here, again, it will be seen—and the fact is important enough to bear a good deal of repetition—that while the blood in the veins is backed up toward the smaller vessels at the periphery, because its exit is impeded or stopped, so the blood in the arteries is driven forward in them until the pressure in the smaller vessels is great enough to resist the increased pressure from without; and thus it happens that in both systems the quantity of blood is greater in proportion in the smaller vessels than in the larger—a condition which is often found in many morbid processes.

It will finally have been noticed that under the pressure the injection of the disk—that is to say, its natural rosy color which is caused by the more minute vessels—becomes paler and paler, until it assumes a dead and even whiteness; and it will be soon seen by the observer that it requires a considerable degree of pressure to produce this or any effect upon the color of the disk—or, in other words, that the larger vessels respond much more quickly to the pressure than the smaller ones.

To recapitulate, we find, then, that the principal changes which have occurred are:

(1.) Pulsations in the vessels.

(2.) A change in the calibre of the vessels.

(3.) A modification in the brilliancy and diameter of the light-streak.

(4.) A change in the color of the blood.

(5.) A change in the vascularity and color of the disk.

As these changes are general in character, and as they are not only the physical expression of many morbid processes, but oftentimes the key to their character and the explanation of their nature, they deserve to be studied somewhat in detail.

Changes in the Diameter and Length of the Vessels.—Changes in the diameter of the vessels may extend over the entire system, arterial as well as venous, or they may be limited to a single vessel or even to a part of a vessel. Thus, both the arteries and veins may be increased or lessened in diameter, or the veins be decreased in width and the arteries remain normal, or, *vice versa*, the veins may be increased in width and the arteries become narrower, which is what usually occurs.

This change in the calibre of the vessel may be due either to a real change in the horizontal or perpendicular diameter of a vessel—that is, to an actual distention or contraction of the walls—or it may be due simply to a change in the form of a vessel. Thus, a vessel may appear to be increased in calibre because it is flatter, the curvature less, and the light-streak broader. On the other hand, where the perpendicular diameter is increased at the expense of the horizontal, the vessel may appear less wide and the light-streak narrower. The former is much more common than the latter, especially in the veins. It has already been explained that, as a rule, the arteries run more superficially than the veins, and that they are smaller, in about the proportion as three is to four. While there is this or even a greater difference in health, which extends to the entire system, it is important to notice that any considerable decrease or increase in a single vessel, and especially a part of a vessel, is usually a sign of disease, except when it occurs at the porus opticus, where, even in a normal eye, a tapering of the arteries as well as of the veins may occur (vol. i., p. 69).

FIG. 1.

Increase in the Diameter of the Veins.—A uniform increase in the diameter of the entire venous system occurs most frequently in

irritation or inflammations of the retina which have affected the walls of the vessel, and which have produced on the subsidence of the inflammation a state of atony, or a distensible condition of the wall; and when this is the case the increase in diameter is usually accompanied by an increase in length, which is shown by the greater tortuosity of the vessels. This condition is represented in the figure, in which all the veins are increased in diameter and somewhat in length, and the arteries reduced not only in diameter but also in length, or at least in their visible length (Fig. 1). Instead of extending to all the

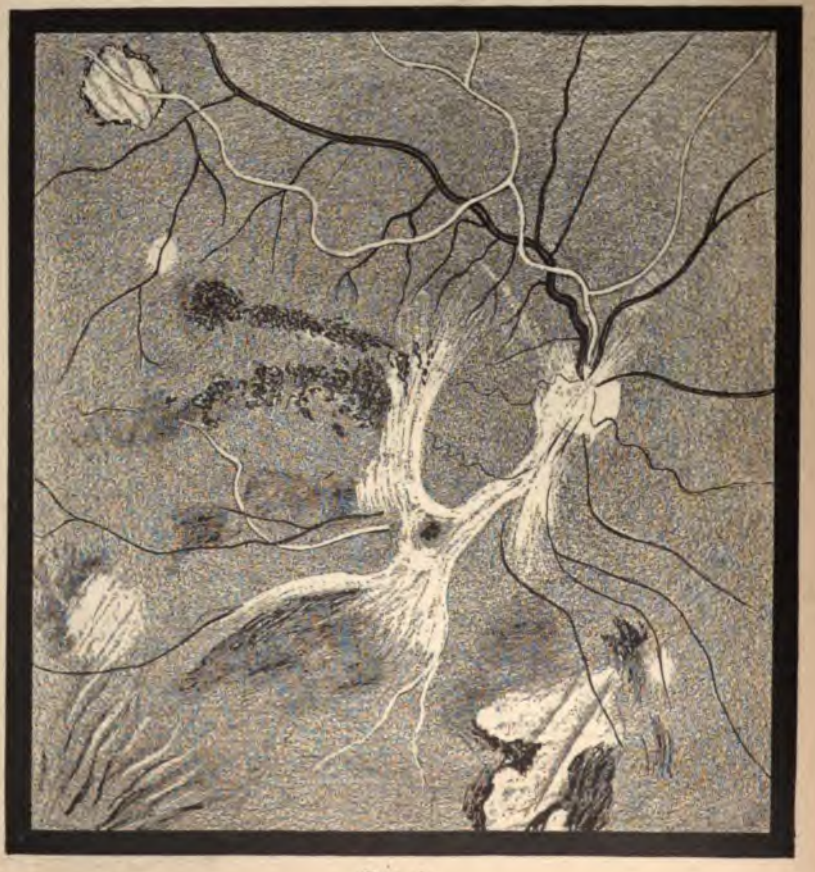

FIG. 2.

veins, this increase may be limited to a part of them, as is shown in a chromo-lithograph of syphilitic retinitis by Liebreich.* It will be seen from the drawing, Fig. 2, that the veins in the upper half of the

* "Atlas d'Ophthalmoscopie," Berlin, 1863; Tab. X., Fig. 1.

picture are much dilated, while those in the lower field are very much reduced in size. The increase in diameter may be limited to a single vein. The most marked instance of this that I have ever seen is

FIG. 3.

represented in Fig. 3. This was the result of a limited inflammation of the retina, and a subsequent dilatation of one of the principal veins. The cause of the trouble was attributed entirely by the patient to an overdose of quinine, which he had administered to himself. The picture was drawn about two weeks after the drug had been taken, and when most of the inflammatory symptoms had subsided. The amount taken was, according to the statement of the patient, "a little more than a teaspoonful of the powder." (See Plate I., Fig. 1.)

This increase in the diameter of the veins is most apt to occur as the result of specific retinitis, though it may occur from any other form of inflammation, especially from splenic retinitis, and is then usually of a marked, sometimes of an enormous, degree.

It is very often temporary in its character, and may vary from day to day, or even in the course of a single day. I have often seen

that congested condition of all the vessels which sometimes accompanies or causes retinal irritation, especially that occurring in middle or later life, and which is so apt to be followed first by retinal and then by cerebral hæmorrhage.

Decrease in the Diameter of the Arteries.—This is much more common than the opposite condition just mentioned, and is due, no doubt, in part to the contractility of the walls, aided as it is by the intraocular pressure upon their external surface. It occurs in the secondary stages of exhaustive diseases, such as the graver forms of fever, in Bright's disease, and in many general troubles where the action of the heart, though weak, is rapid. Here the arteries, besides being reduced in breadth, are also reduced in length, and, from being comparatively wide at the disk, become rapidly smaller in diameter as

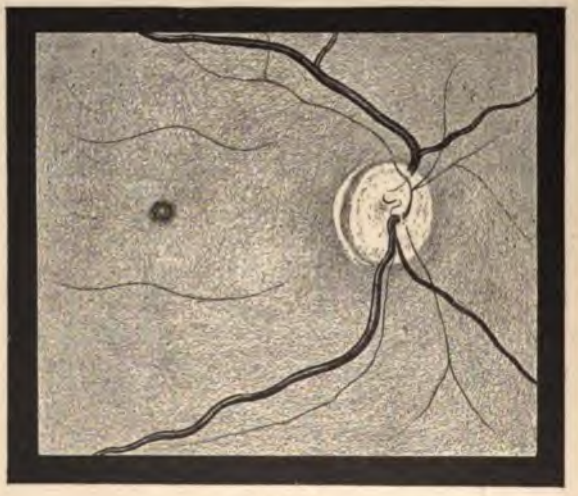

Fig. 4.

they extend into the retina. Hence in some cases they are lost to view much sooner than would seem possible from their comparatively large size at the porus opticus. In embolism, especially when not complete, and in thrombosis and venous obstructions, the arteries, though they are narrower, are so near the disk, increasing in size toward the periphery. This contracted condition of the arteries is very marked in some forms of optic atrophy. Fig. 4 is from Liebreich, Tab. 8, Fig. v, and shows the contraction of the arteries and atrophy of the nerve following embolism of the central artery. It reaches, however, its greatest height, as a rule, in the secondary contractions which follow after inflammations of the retina and optic nerve, as, for

example in the retrogressive changes in choked disk, in Bright's disease, and other forms of retinitis in which the tissue-changes are very marked, and which are followed by complete atrophy of the retina. I have seen cases in the retrogressive stages of inflammation where the veins still maintained a considerable if not normal calibre, and yet where the arteries were so reduced as to be only distinguishable at or very near the disk; and the question at once suggests itself how is it possible for the arteries, in their almost obliterated state, to furnish sufficient blood to produce hæmorrhages of such a degree.

It is certainly curious and at variance with the commonly accepted idea of inflammatory diseases, that, in most if not all of the inflammations of the retina, the arteries, instead of being enlarged, are reduced in size, and instead of being increased in length are shorter than normal, as can be seen by their straightened course. It is only exceptionally that the arteries are enlarged and tortuous. This may, however, occur at times, especially in leucocythemic retinitis, where the arteries are at times enormously dilated and tortuous, as will be seen in Fig. 5. The narrowing of the arteries may extend to all the branches or be confined to a few, or even to a single branch. Thus, in certain

FIG. 5.

forms of retinitis, I have seen a single artery run like a fine and tensely drawn red thread in a perfectly straight course for half the field, and

then resume its normal size, presumably through some anastomosis, though none could be detected.

When the narrowing takes place after inflammatory conditions, it is more marked, as a rule, toward the nerve than at the periphery, so that the vessels, although narrower at the start, carry a uniform diameter for a longer distance into the retina.

Narrowing of the arteries is often, if not usually, met with in glaucoma, and this is what would be naturally expected wherever the intraocular pressure was increased upon the outer surface of the walls. But what does appear at first sight a little curious is, that the arteries are either of the normal size or are reduced in diameter where the intraocular tension is diminished.

In debilitating diseases, where the action of the heart is reduced, the arteries are apt to be smaller than normal, as in anæmia and chlorosis and kindred diseases.

Increase in Length of Vessels.—Remarkable as is the increase in breadth of the vessels, amounting, as it sometimes does, to double or treble the ordinary diameter, the increase in length is even more so. For, besides the tortuosity which we commonly see, and which in itself may be of a very high degree, the vessels sometimes double upon themselves in long sinuous loops, by which the vessel is greatly increased in length, and consequently the quantity of blood circulating in the eye at a given time must be also enormously increased, and this, too, sometimes without the slightest increase in the intraocular pressure, or any impairment of the function of the organ.

To what this extension in the length of a vessel is due is sometimes difficult to determine. It would naturally be supposed that it was caused by some direct impediment to the circulation; but in point of fact it is not, and it often exists to a marked degree where, on section, there are no evidences of pressure upon the vessels, or any signs of strangulation or decrease in the lumen of the vessel. It may occur upon all the vessels, or be limited to a single vessel or part of a vessel. It may be present or absent in one and the same disease, or appear as the first or last symptom of that disease. It would seem, then, as if the trouble must be due to some inherent condition of the wall of the vessel, which caused its elements to yield to the pressure of the circulation in a longitudinal as well as transverse direction.

This extension in length of a vessel is usually accompanied by a contemporaneous extension in width, but not always, since the transverse diameter may remain either unchanged or even become less than normal. Thus, in retrogressive changes after violent inflammatory processes, the secondary contraction may take place to a greater degree

in the calibre of the vessel than in its length. It may also occur in some low-graded and chronic inflammations, such as are present in certain forms of neuro-retinitis, or especially of chorio-retinitis.

As these secondary contractions usually occur with more force in the larger vessels near the disk, they lose thereby what mechanics call the "natural diminish," and carry a uniform diameter farther into the retina, which may indeed be greater toward the periphery than near the nerve.

This behavior and appearance of the vessels, together with evidences of vasculitis and perivasculitis, to be mentioned later, are oftentimes a useful guide in determining whether a present attack has been preceded by earlier ones, especially if the abnormal appearances exist in both eyes and there is complaint only in regard to one.

Thus we see, to recapitulate, that in some conditions both sets of vessels are increased in size at the same time, and that consequently the amount of blood in the retina is increased. Secondly, that both sets of vessels may be decreased in size, and, consequently, the amount of blood in the retina is decreased. Thirdly, that in some of the most frequently occurring inflammatory conditions the arterial supply is reduced and the amount of venous blood is increased. Fourthly, and this is the rarest of all, the arterial blood is increased over that of the venous.

The decrease in arterial blood is shown not only by a decrease in diameter, but also by a decrease in length of the vessel, as is made apparent by the fact that the arteries are straighter in their course, and that they can not be followed in the retina as far as is usually the case; and that sometimes they are entirely lost to view, when, under normal conditions, they would not only be perceptible but of considerable size. On the other hand, the increase in the quantity of venous blood is shown not only by the greater breadth of the vessel but also by its greater length, which is expressed by the increased tortuosity of the vessel and by the increased visibility of minute vessels.

Variations in the Color of the Blood.—Jaeger has expressed the opinion that there is but very little variation in what may be called the standard color of venous and arterial blood under physiological conditions, and that the slight differences which occur are not due to the color of the blood itself, but to the conditions under which the blood-column is observed: that is to say, they are due, when they occur, to the quantity and quality of the light, the size of the pupil, the age of the patient, the transparency of the walls, the dimensions of the horizontal and perpendicular diameters of the vessel, and the

proportions which these bear to each other, as well as the enlargement under which they are seen.

The writer is of the opinion, on the contrary, that besides all these influences, which certainly have a decided effect, there is even in health a marked difference in different individuals in the color of the blood itself, and that this difference should not be lost sight of in estimating the degrees of color in disease. Thus, I have been led to believe that the blood in some brunettes was darker than the average physiological blood, and that such persons possessed not only a larger amount of fixed pigment in their organization, but that they had a capacity for producing a larger quantity and a deeper shade of coloring-matter for the circulating fluid.

I have, for example, now under my observation a pronounced brunette, with a very dark olive skin, in whom this effect of a darker and richer color in the retinal vessels is present to a marked degree. The person is in perfect health, and the pigmentation of the choroid, though rich, is by no means excessive; and yet the blood in the retinal arteries is of a deep, clear red, approaching a maroon, while that in the veins is of a blackish red. This would go to show that the deeper color of the complexion is not simply due in these cases to the pigment in the skin, but is caused, in some degree at least, by the color of the blood circulating in the capillaries and the smaller vessels. This would explain why it is that some of these people become so deathly pale under great emotion or in a fainting turn. So, too, with blondes, in whom it is but natural to believe that with a lighter complexion there may be also a lighter-colored blood.

Be this as it may in health, there can be no doubt but that in many pathological conditions, marked variations occur in the color of the blood: that is to say, the blood may not only become of a lighter or darker shade of red, but may, to a certain extent at least, alter its color.

When the blood becomes of a lighter or darker shade of red, the variation usually affects both systems, arteries as well as veins, so that, although a definite difference in tint is present, the relative proportion in shade between arterial and venous blood is still maintained; and if the arteries are of a darker color than usual, the veins are still proportionately darker than the arteries.

This, however, is not always the case, as it frequently occurs that the change in shade is limited to one set of vessels, so that the veins may become lighter or darker and the arteries remain the same, or the arteries may be darker or lighter and the veins remain the same: that is to say, the blood in the viens may become like arterial blood,

or the arterial grows darker like venous, so that the color in the two systems is equalized.

The greater the perpendicular diameter of the vessel the darker the color; and, *vice versa*, the less this is—or, in other words, the flatter the vessels—the lighter the color. This is particularly well marked on the arteries, especially where the intraocular pressure is increased, or where the action of the heart is feeble: that is to say, wherever the intraocular pressure is proportionately greater than the pressure of the blood-column. The quicker the current, the stronger the illumination, the wider the pupil, and the greater the enlargement under which the vessels are seen, the lighter will they appear. The more the light is absorbed by the fundus of the eye, or by the tissue surrounding the vessel, the darker the color, and the more light the fundus reflects, the lighter the color of the vessels, this being due probably in the former case to the reduced and in the latter to the increased amount of diffuse reflection within the eyeball; and here it may be observed that ophthalmologists have not usually paid any attention to the existence of this kind of light and its effect upon the fundus, though it must be present in all cases, and in some to a considerable degree. There is an exception to the rule that with the reduction of light the vessels look darker, which I can not well account for, and that is, that where the vessels, especially the veins, are seen through a diffuse opacity, or a light membrane in the central parts of the vitreous, they appear lighter, and not, as would be expected, darker.

I have thought that perhaps this might be due to a slight infiltration into the lymphoid spaces of the vessels which was a concomitant of the existing trouble. That this, however, is not always so is shown from the fact that if the patient's eye is turned as far as possible to one side, and the more peripheral parts of the same vessel observed, the lighter shade vanishes and the vessel comes out in its normal color. I have seen the same thing in serous iritis and low stages of choroiditis of a glaucomatous type.

As a rule it is in troubles of the general system that both sets of vessels are affected by a variation in tint which in some cases is very marked. Thus, all the retinal vessels are of a darker red color in those conditions where the blood becomes thicker from an increase in its solid contents or from a removal of its fluid elements, as for example in those diseases which are accompanied by excessive discharges from the bowels, as after exhaustive attacks of dysentery, diarrhœa, or cholera (Graefe, Jaeger).

The most marked example which could possibly be given of an increase in color of the blood in all the vessels is found in those rare

cases known as cyanosis bulbi. Here, from mixture of arterial and venous blood, from imperfect oxygenation and from retardation of the current, the blood in the arteries is usually of a dark maroon red, similar to what is sometimes seen in the veins, while the veins have a purple or violet tinge. On the other hand, all the retinal vessels appear of a lighter color when the blood is deprived of its normal amount of solid constituents, or where there is a change in their condition or the proportions under which they usually occur. Thus, the blood is of a lighter red in both the veins and arteries in anæmia, leucocythæmia, in some cases of chlorosis, and Graves's disease, or wherever a condition of anæmia or hydræmia is produced. In some cases of splenic retinitis the change in the color of the blood is very marked, assuming a light orange, a rose pink, or a grayish pink, instead of the ordinary crimson red.

In pernicious anæmia the blood in both the veins and arteries is in some cases much reduced in color—so much so that in the arteries it is often of a pale yellow or straw color, and in the veins much lighter than ordinary arterial blood. In one case, which I saw at St. Luke's Hospital, the arteries looked like yellowish-white bands, and the veins, although of larger size, had much the same appearance. In some cases, as will be seen later, the change is only in the veins. In other cases, again, the normal discrepancy is maintained. It would be interesting to know what was the cause of this difference in one and the same disease.

There are other cases where, instead of existing in both systems, the change occurs, or at least predominates, only in one. This change, when it occurs, does so principally in the veins, so that the color of one set of vessels approaches that of the other: that is to say, the venous blood becomes lighter and of a purer red, so as to resemble arterial blood; while on the other hand, which is much more rare, the arterial blood becomes darker and approaches or equalizes the color of venous blood. A darker color of the venous blood, with a normal or even lighter color in the arteries, is found in febrile diseases of a high grade, especially where the action of the heart, though rapid, is weak. This increased color in the veins is no doubt due to the fact that the current in the veins is less rapid, and that here as elsewhere the consumption of the oxygen is greater, owing to the greater time that the blood is in contact with the walls of the veins, and the more wasting character of the tissue-changes which are going on.

Thus, this disproportionately darker blood occurs frequently in certain stages of pneumonia and pleurisy, and in some cases of asthma and emphysema. Moreover, in the later stages of these inflammatory

troubles the arteries become lighter than normal, which is probably due to a change in the blood itself, especially to a reduction in the quantity or a change in the form of the red corpuscles. According to Jaeger, the veins become darker in asphyxia.

They are also darker in some cases of inflammation of the retina and from diseases in the orbit; and that the trouble is then purely local can be seen from the fact that the increase in color does not exist in the veins of the other eye.

A lighter color of the venous blood, while that of the arteries remains the same, is found in certain forms of anæmia, chlorosis, and chronic forms of disease of a debilitating character. It occurs very commonly in leucocythæmia, anæmia, and kindred diseases. It is also found in certain forms of retinitis in which there is a diffuse œdema into the surrounding tissue; and that in these cases it is due to local causes is shown by the fact that in the fellow-eye, in which there is no trouble, there is still the normal discrepancy between arterial and venous blood. I have a case now under observation in which the veins are of the same light shade as the arteries, and can only be told from them by their larger size and more tortuous course. There is evidently a mild state of diffuse œdema into the retinal tissue, especially along the course of the veins, and there is no light streak upon the veins or arteries. There is no pulsation of the veins upon pressure, and only a slight one upon the arteries, which taper at the disk, as do also the veins. The nerve is pale and œdematous, with indistinct outlines; vision is reduced to counting fingers at one foot. There is evidently a disturbance in the circulation, due perhaps to embolism, or more probably to thrombosis. The fellow-eye is not affected in the least, and the normal discrepancy in color between arteries and veins is maintained.

In ordinary anæmia and in pernicious anæmia also—a disease where the normal proportion of the red to the white corpuscles is supposed to be maintained—this reduction in the color in the veins is often observed, and a satisfactory explanation of this occurrence in disorders of a general character is difficult to arrive at. Jaeger asks the question if it can not be due to the fact that for some reason, not yet understood, the hæmaglobin in the red corpuscles refuses to part with the oxygen which it holds in combination, and thus the blood remains arterial in quality in the veins. I have, on the other hand, been inclined, at times at least, to look upon this lighter color of the veins as a purely local manifestation produced within the eye itself, due to the effect occasioned by an altered state of the blood upon the walls of the vessels and upon the surrounding transparent

tissue of the retina, or to an altered and less transparent condition of the fluid in the perivascular spaces. That it is not due to an increased quantity of white corpuscles is shown by the fact that it is often present where the normal proportion between white and red corpuscles is maintained, as is shown by the microscope.

In the atrophic stage which follows the more violent forms of inflammation of the retina, such as specific retinitis, retinitis hæmorrhagica, splenic retinitis, retinitis albuminurica, and that which follows choked disk, or, in a word, any form of inflammation in which the vessels have participated, not only do the veins become smaller, but the color approaches that of the arteries, which shows that the transparency of the wall has suffered; and that the effect is really due to the condition of the wall is shown by the white lines along the sides of the larger vessels, and by the hyperplasia of the connective tissue which surrounds them. Also, in what looks like pure and simple white atrophy of the optic nerve, and where there is no sign of any retinitis, the color of the veins is changed to a lighter hue, showing that the wall of the vessel has participated in the degeneration, or has suffered from some low form of inflammation; so, too, in the sclerosis of the vessel wall which accompanies retinitis pigmentosa and the secondary stages of amblyopia potatorum, and also in some forms of atrophy from spinal disease.

A predominating darker color in the arterial blood, so that the discrepancy between arterial and venous blood is equalized, is observed, according to Jaeger, in some forms of stasis hyperæmia. This is a change which I can not remember to have noticed in any form of eye-disease except in cyanosis.

Dr. Heyl has published, in the "Transactions of the American Ophthalmological Society" (1880, p. 54) an interesting case, in which the color in the veins was the same as that in the arteries, and which he has called intraocular lipæmia, under the assumption that the color was due to emulsified fat. "The patient was weak and much emaciated, and, according to the history of the case, was suffering from diabetes mellitus to a marked degree. An examination showed the white corpuscles to be as 1 is to 84, while that in health is as 1 to 350 or 400. On pricking the finger a drop of blood would escape, having very much the color of a piece of pink coral, followed by a thick, whitish fluid of the color and consistency of an oil emulsion. On examining this with a low power of 250 diameters a few blood corpuscles could be seen upon a dim, misty background, barely discernible. Examination with a power of about 600 diameters showed the granular nature of this background, and examina-

tions in various ways left no doubt but that it was fat in a finely divided state.

"The ophthalmoscopic examination revealed the following particulars: The fundus was free from the existence or pre-existence of inflammation; the margin of the nerve was well defined, and there were no retinal lesions or hæmorrhagic clots. The vessels were all apparently of the same color, which may be described as a light salmon color. When the light was thrown directly upon the vessels, the arteries could not be distinguished from the veins; by rotating the handle of the mirror so as to deflect the light, a dark axis could be seen in the large venous branches. Another curious phenomenon was that the vessels appeared to have about twice the normal calibre.

"This condition, to which the name lipæmia retinalis may be given, is therefore characterized by the light salmon color of the blood which is contained in the retinal vessels, and by the increased diameter of these vessels, and possibly by the very light color of the fundus oculi—these appearances being due to the presence of fat in abnormal amount in the serum of the blood."

If the appearances here described were really due to finely divided fat in the blood, it would offer not only an explanation of the color in the vessels, but also give evidence for the solution of the cause of the hæmorrhages which sometimes occur in other diseases in which fat is present in the blood, and which then might be attributed to fat emboli; for in a circulation so fine as that of the smaller vessels of the eye the emulsified fat might well combine into fat-globules, and these, in the retarded circulation, especially of the veins, and the slower movement of the plasmic current, might so block the vessels as to occasion a rupture of the wall. It might also explain the peculiar light color noticed in some cases of pernicious anæmia and not in others, and account by its presence or non-presence for the difference which we see in the color of the veins, not only in this disease but in many others. The fact that fat was found in the blood on puncture of the finger, and that the blood was of a milky hue when taken from this locality, is very strong, indeed the strongest, evidence that it existed everywhere in the circulation, and consequently in the retinal vessels. But still it must not be forgotten that in this case the blood, if not really leukæmic was very nearly so; moreover, it must be noticed also that all the opthalmoscopic signs were those which may occur in pure splenic retinitis, for in this disease we sometimes see an enormous dilatation of both veins and arteries, so that the vessels are twice or even three times as large as normal vessels. Again, the general color of the fundus is not always of an orange hue but may

be a pale pink. I have at this moment a case of splenic retinitis under observation at St. Luke's Hospital, in which the fundus is of a pale grayish pink, the vessels considerably enlarged, and the arteries larger than the veins; the color of the blood in the vessels lighter than normal, and almost identical in shade in the two systems.

It must be remembered too that we sometimes see a reduction in the color of the veins so as to equalize that of the arteries in purely intraocular disease, especially from œdema into the retina or perivascular spaces, where the trouble is local and confined to a single eye.

Still, the observation is most interesting and deserves a careful investigation, not only for the supposed condition of lipæmia, but in regard to the possible connection of a fatty condition of the blood with other still unexplained processes and appearances, especially those of the nature of embolism and thrombosis.

Changes in the Light-Streak upon the Centre of the Retinal Vessel.—These may relate to variations in breadth, color, intensity, and uniformity. The breadth of the reflex varies often a good deal, not only in health but in morbid conditions. This is due in great part to the differences in proportion between the horizontal and perpendicular diameter of the vessel. It must here be borne in mind that the lumen of a retinal vessel on section is not a circle but an ellipse. This is due to the fact that the vessels are not completely filled with blood, and the veins less so than the arteries. To an inattentive observer this ellipsoidal character of the vessels is not very apparent. But when the attention has been once called to it the vessels seem to vary a good deal in their rotundity and flatness, according as they are in a state of congestion or emptiness. Thus, in some cases they appear to be tense and round, and in others flat and ribbon-like, and this is especially true of the veins. From this we get the general rule that the flatter the vessels, the broader the light-streak; and the rounder it is, the narrower the reflex. This rule has, however, many exceptions, which would appear to be due to the color and composition of the blood and the transparency of the surrounding tissue, for the darker the blood and the less transparent the wall and enveloping tissue, the narrower, or at least the less conspicuous, does the light-streak appear.

The variations in the color of the reflex are not, as a rule, marked, although there are cases which show a considerable departure in shade at least from the normal color, which may vary from a pale yellowish straw color to a yellowish white or even to a yellowish pink, especially in the veins. The light-streak, as a rule, appears lighter in color and more uniform in character in the arteries than in the veins; and it is more constant in its appearance and less interrupted

in its course in the former than in the latter. Its borders too are more clearly cut, and it can usually be traced farther and upon finer vessels in the arteries than in the veins.

Hence we get the general rule that the darker the blood the less brilliant the light-streak, which is exemplified in most of the general diseases in which we have just seen that the color of the blood becomes darker in the retinal vessels.

As the stronger reflex in the arteries is mostly due to the lighter color of the blood, when the blood in the veins becomes lighter, so as to resemble arterial blood, the light-streak on the veins also becomes more brilliant and equalizes that of the arteries. When, on the contrary, the arteries become darker, so as to resemble venous blood, the brilliancy of the light-streak is reduced, so that the two are equalized.

In those cases in which, as we have seen, the blood becomes darker in the veins and at the same time lighter in the arteries, the reflex is then darker on the veins and lighter on the arteries than normal, and the discrepancy in the two is then at its highest. A marked example of this is seen in the effects of intraocular pressure, as, for instance, in glaucoma. Here it can be often seen that the pressure increases the roundness of the veins by impeding the exit of the blood from the eye, which, from the retardation of the current, becomes of a darker color. The light-streak is consequently reduced both in size and brilliancy. As, on the other hand, the blood is impeded from entering the eye, the arteries are less full, and therefore flatter than normal, the color of the blood lighter, and hence the light-streak is both stronger and broader. This may also occur in any form of disease where the arteries are reduced and the veins increased in size.

The intensity of the light-streak varies more than its actual color, for, while it is usually of a subdued hue, it may rise to almost a silvery brilliancy. When this is the case it is usually due to light, feathery particles or shreds of connective tissue, which reflect the light from the summit of the vessel. That this effect is due to the wall is shown by the fact that the larger vessels under pressure still remain visible at the porus opticus after the blood has been expelled from them, and betray their presence not only from the white, narrow lines which mark their lateral boundaries, but also by a fine, feathery reflex which extends across the entire diameter of the vessel.

The brilliancy of the reflex must also be affected—either increased, decreased, or annulled, by the comparative degree of transparency or opaqueness of the wall.

An altered index of refraction of the wall of the vessel would also have its effect, did such alteration occur; but, although there is some

reason to suppose that such a thing may take place in some diseases, notably in Bright's disease, actual proof of its occurrence has not as yet been furnished.

Any disturbance in the media lying in front of the retina naturally interferes with a perfect illumination of the bottom of the eye, and, inasmuch as the details of the fundus will be more or less obscured, the light-streak will be proportionately affected. Still, when even, on this account, only a moderately distinct view of the fundus can be had, the light-streak on the retinal vessels will be comparatively well marked, allowance being made for the general haziness of the media. But, on the other hand, though the media in front may be perfectly clear, the slightest œdema into the retina will cause the light-streak to be much diminished, or even to vanish entirely. And this takes place even if the œdema is confined to the immediate neighborhood of the vessel, and occupies, so to speak, only the perivascular space. Should the œdema be of any extent, the vessels have no central streak whatever, but present a uniform red appearance. This is markedly the case where the œdema is very pronounced, as it sometimes is in embolism or injuries from blows on the eye, or in some forms of acute retinitis.

Without laying too much stress upon it, I have been led to look on this absence or presence of the light-streak as a useful and important sign in the differential diagnosis between troubles in the superlying media, especially in the vitreous, and haziness in the retina itself.

In some cases of overworked eyes, where there has been no other ophthalmoscopic sign, I have often noticed a decided diminution in the brilliancy and size of the light-streak, or even a total want of it, especially in the veins. I have attributed this to some slight impediment in the circulation, and a consequent transudation of serum into the perivascular spaces or tissue immediately surrounding the vessels. I have seen precisely the same thing in patients who have been suffering from some affection of the heart, though I have never been able to trace this, as Becker does, to the single condition of aortic insufficiency. Nor have I, as he does, attributed this reduction in size and brilliancy to the reduced calibre of the vessel; for I have seen cases where the light-streak was entirely absent, even where the vessels were abnormally distended, and have then thought it due to the abnormal condition of the blood, or of the walls of the vessels or surrounding tissues, which interfered with their usual transparency.

I have noticed, too, this disappearance of the light-streak in one or two instances of what afterward proved to be commencing separation of the retina, several days before the actual separation took place,

the separation corresponding pretty nearly with the tracts of the retina where the light-streak was absent. In one of these cases the retina readjusted itself and remained applied for some three weeks, and then finally gave way. In another case, where the line of demarkation between where the light-streak was present and where it was not, in all the lower vessels, was well marked, and where, too, there was a corresponding limitation of the field of vision, no separation of the retina took place, and the œdema around the vessels, or what was supposed to be such from the absence of the light-streak, gradually passed off.

In actual separation of the retina the light-streak is wanting.

I have also observed that the light-streak is absent, in different degrees, however, in many conditions in which the vessels themselves seem to have suffered from inflammatory action, especially in perivasculitis, or perhaps inflammation of the wall itself, and where secondary atrophy of the tissue has taken place, with hypertrophy of the connective-tissue elements. Thus the effect is often very pronounced in the secondary or atrophic stage of retinitis albuminurica, in those cases where the vessels themselves seem to have been involved, and finally to have become reduced slightly in size. In these cases the light-streak is often very much diminished, or even entirely wanting.

Precisely the same thing takes place in some cases of retinitis pigmentosa, and oftentimes in the earlier stages of the disease where the vessels are just beginning to suffer a reduction in size, and where as yet the accumulation of pigment in the retina has not taken place to any considerable degree, or even where this is entirely wanting. Moreover, in these cases where the light-streak is absent, it is not universally so—that is, in all parts of the fundus—for it may still be present in all its normal dimensions and brilliancy in some places, while in others it is much reduced or even wholly absent.

Precisely the same thing occurs oftentimes in those curious cases of retino-choroidal atrophy, occurring especially in old people with atheromatous arteries, and where, in different parts of the fundus, but usually near the macula, well-defined regions exist, which show an absorption of the choroidal epithelium, capillary layer, and pigment of the stroma, to such a degree that the larger vessels seem to be dissected out and to stand in vivid contrast with the adjoining parts of the fundus which still preserve a normal appearance.

I would say here that I do not allude at all to the well-known atrophic patches seen in choroiditis disseminata, or areolaris, where the retina itself is not usually much affected, but to that condition which

Jaeger has described (and of which he has given two admirable drawings*) as atrophy of the pigment layer of the choroid.

It would appear that this diagnosis is not correct, or at least not comprehensive enough, since simple atrophy of the choroidal pigment layer never in itself produces any great disturbance in vision, as we see in posterior staphyloma, where not only the pigment-layer but almost the entire choroidal tissue has been absorbed. And in all these cases of ordinary choroidal disease, we very rarely if ever see scotoma —never, in fact, unless the retina is affected. But in the cases to which I allude, and in those which Jaeger has figured, we find great disturbances of vision and well-marked scotoma, showing that the retina is implicated, and it is in such cases that directly over and in the immediate vicinity of these circumscribed patches, the light-streak of the retinal vessels is often completely wanting. Through the kindness of the late Dr. Althof, I was enabled to present a beautifully marked example of this to the New York Ophthalmological Society. In this case, that of an old woman of some seventy years, the light-streak could be seen perfectly well in some places, while in those parts of the fundus corresponding to the atrophic condition of the patches it was either very much diminished or entirely wanting. I have also seen this want of the light-streak in some cases, chiefly among old people, where there was no other ophthalmoscopic sign, but where, from other symptoms, there was reason to believe that the vessels of the general circulation were more or less affected with atheromatous or amyloid degeneration.

So, too, in cases of what looks like simple atrophy of the disk, with shallow excavation, I have noticed this diminution or want of the light-streak, and a closer inspection then always reveals the fact that the fundus has a slightly hazy appearance with delicate changes in the choroid. I have been led to believe, in such cases, that the retina itself was affected, and that the nerve-fibre layer especially participated in the trouble which was so plainly marked in the united nerve-bundles in the disk.

Whether in the foregoing and similar cases the above-mentioned absence of the light-streak be due to an abnormal thickening of the vessel-wall itself or that of the adventitia, or to a slightly œdematous condition of the tissue immediately surrounding the vessel, I am unable to determine, but I believe that at times it is due to all of them.

Changes in the Walls of the Vessels.—Decrease in the transparency of the walls varies from the merest trace to a complete opacity

* "Hand-Atlas," Taf. xxii., Fig. 97, text p. 165.

of the tissue. It depends usually upon fatty degeneration, sclerosis, perivasculitis, or chalky infiltration.

These disturbances extend into the wall of the vessel, either in a regular or irregular manner, and show themselves either in a granular or *punctate* form, or as longer or shorter stripes which may follow either the length or breadth of the vessel. They have commonly a yellowish-white or greenish-white appearance, according to the amount of connective tissue they contain, or they may be of a reddish tinge, and this may be carried so far as to suggest the idea that these opacities are themselves organized. They may occur either as a general disturbance which affects all the vessels, or as a sign of a process which is limited to certain vessels or parts of vessels.

They usually occur on both sides of the vessel, but oftentimes only on one, and as a rule they exist most frequently and are most strongly developed on the larger vessel upon and near the disk.

Through this want of transparency the walls of the vessels are rendered more visible, and then often show themselves as yellowish-white and delicately-marked bands which line each side of the blood-column. These sometimes extend across the diameter of the vessel, which then becomes visible for its entire breadth. Usually, however, a fine red line is seen upon the apex of the curvature, but that even here the entire breadth of the wall is affected can be told by pressing the blood out of the vessel, when the wall becomes visible for its entire width.

Sclerosis of the Walls.—This consists in a thickening of the tunica propia, by which, according to Leber, it becomes more homogeneous, of a greater index of refraction, and loses, to a greater or less degree, or even entirely, its structural elements. The process has a similarity in appearance with amyloid degeneration, but differs from it in lacking the characteristic reaction. It attacks principally the arteries and capillaries, and to a lesser degree the veins, and causes what is known as sclerotic thickening of the walls by which the lumen of the vessel is much reduced or obliterated. As the hyaline substance which encroaches upon the lumen of the vessel is perfectly transparent, the walls themselves are not rendered any more visible than before the process took place, and the existence of the trouble is only to be recognized with the mirror by the reduction of the diameter of the red blood-column.

Sclerosis of the vessels may occur in any form of retinal inflammation, notably in nephritic retinitis, and more notably still in that form of retinitis which is accompanied by pigment degeneration usually known as retinitis pigmentosa, whether of the typical or a-typical

form. Here both the arteries and veins are affected, so that a gradual decrease in the lumen of the vessel takes place through the hyaline thickening of the wall, by which the blood-column is gradually reduced in size until it becomes thread-like in its dimensions or even imperceptible.

This form of sclerosis, or hyaline thickening of the wall, may account for an appearance with the mirror which otherwise would be somewhat difficult to explain. We often see in some forms of localized inflammation of the retina, and especially in chorio-retinitis, that the blood-column of a vessel which happens to cross or run alongside of the inflamed portion will become markedly narrower as soon as it arrives at the spot, and will regain its normal width again as soon as it has passed the affected district. As the surface of the vessel is not whitened at all, and as there are no signs of perivasculitis, I have been inclined to look upon such reductions in the diameter of the blood-column as due to a reduction in the lumen of the vessel, especially as no anastomosis could be detected.

Besides this transparent form of sclerosis, there is a similar process in which the resulting tissue is non-transparent, and which has been described by Jacobson as affecting the walls of the vessels, and giving to them with the ophthalmoscope the appearance of branching white lines or cords. The appearances described by Jacobson were observed in a young man in whom there was a tumor of the optic nerve and orbit of the left eye. The ophthalmoscopic picture was principally remarkable for the peculiar appearance of the retinal vessels and the changes which were present upon the disk. Those parts of the retinal vessels which had become whitened usually lay between two portions of the vessel in such a way that there could be no doubt but that the opaque and non-opaque parts were connected by a continuous current of blood. Only the column of blood in the whitened parts was as much smaller as the opaque walls were broader, so that at times it required very careful observation to recognize the thin red line which represented the blood-column in the centre of the vessel. The eye was enucleated and given to Prof. Recklinghausen for microscopic examination, which gave, so far as the vessels were concerned, the following results:

The entire wall, in the case of the larger vessels upon the disk, was sclerosed. The lumen of the vessels, on account of the increased thickness of their walls, was very much narrowed, and here and there as it appeared, entirely obliterated. The branching white cords, representing the retinal vessels, were due also to sclerosis of the walls of the same character as that which affected the vessels on the disk.

Jacobson does not think this form of sclerosis of the vessels diagnostic of any particular form of retinal disease, but common to inflammatory conditions affecting the optic nerve or retina. He also believes that it may exist as a disease of the vessels themselves without any connection with any other pathological process.*

That a sclerosed and whitened condition of the vessels of the retina may follow as the result of inflammations about the eye, as, for example, in cellulitis of the orbit and erysipelas of the face and scalp, I think, admits of little doubt. Thus, I believe that the whitened condition of the vessels, as described by Jaeger, Coggin, Knapp, and many others, is in all cases due to vasculitis. Dr. Knapp is, however, of the opinion that, in his case and others, the conversion of the vessels into white cords was not the result of an inflammatory thickening of the coats of the vessels, but an organization of thrombi, caused by retarded circulation brought about by constriction of the vessels by pressure.† The evidence, however, against this view, furnished by the history and ophthalmoscopic appearances as related by Dr. Knapp and others, is very strong, and is in favor of an inflammation of the wall itself and the subsequent conversion of the vessel and its entire contents into connective tissue.

Perivasculitis.—This consists of a hyperplasia of the connective-tissue elements of the external portions of the wall of the vessel (the adventitia) and the surrounding tissue. Nagel's classical description of this affection, which furnishes one of the most striking of ophthalmoscopic pictures, is as follows:

In both eyes of a young man who had good central sight, but a defect in the field of vision, all the retinal arteries and their branches appeared as if transformed into white strings. One would at first sight be led to suppose that all the arteries were empty, and that this general absence of all blood was due to a plugging or compression of the central artery. A little further examination showed, however, that this was not the case, and that the circulation went on undisturbed in the whitened arteries, and that the white color was due simply to a change in the walls of the arteries. The larger branches showed in the centre of the white stripes a fine red line, and some few arterial twigs still preserved their natural red color.

The veins, however, acted differently, and the larger ones, with the exception of a general reduction in size, and a trifling unevenness in calibre, remained normal; and it was only toward the periphery that the single branches with their finer ramifications were changed

* "Archiv für Ophth.," Band X., ab. 2, p. 73.

† "Archives of Ophthalmology," vol. xiii., Plate I, p. 102.

into white strings, or rather tubes. The pathological change, therefore, seemed to be continued from the arteries over on to the veins. The retinal tissue was, in particular districts, which corresponded pretty closely to the defects in the field, untransparent and disturbed, and it was only toward and beyond the equator that the choroid could be seen with the normal clearness. Numerous punctiform hæmorrhages were scattered about in the diseased parts of the retina, in close groups about the offshoots of the single branches of the veins which were more numerous than normal.

The optic nerve was covered by a protruding mass of glittering white color, which occupied the middle of the papilla. Upon the disk of the right eye there was a red spot, which, upon closer examination, proved to be composed of striæ and minute dots, closely arranged together. These were evidently vessels of new formation in the surrounding mass.

Under moderate compression of the globe the red color of the spot faded away, and returned again on relaxation of the pressure. On the left disk, where the vessels of new formation existed in a less degree, the single loops could be seen distinctly. The growth of these vessels could be followed for weeks at a time. The progress of the disease was a gradual but progressive one. The defect in the field of vision increased very gradually. The central vision was good, except that in the eye, where the defect in the field extended close up to the macula lutea, it was somewhat affected. The general health was good and the heart sound, and there was no sign of any commencing central lesion.

The disease was looked upon by Nagel as a slowly progressing and inflammatory process, which, beginning as an hypertrophy of the connective tissue of the walls, extended from them to the connective-tissue elements of the retina, and which from the arteries passed through the capillary system to the veins.

In conclusion, Nagel points to the analogy which exists between this condition and certain paralytic affections of the nerve-centres, and which have been shown to have as their point of origin a similar morbid process in the walls of the vessels; and especially does he refer to a case of gray degeneration of the brain and spinal column reported by Rindfleisch, and to a case of acute insanity by Leidesdorf.*

It is certainly interesting to bear in mind in this connection, though I have never seen it stated elsewhere, that in certain forms of atrophy of the optic nerve, both of the gray and white varieties, we certainly do see a change in the walls of the retinal vessels, as indeed

* Zehender, 1864, p. 394.

we do also in some forms of amblyopia which have hitherto been set down as due to retro-bulbar neuritis, as, for example, in amblyopia potatorum. Whether the trouble originates as a disease of the wall, or whether the changes in the wall are secondary, is a matter still to be determined, but it is certainly one of great interest in connection with the nourishment and atrophy of nervous tissues.

Nagel closes his description of the case with the opinion that the true nature of the disease will have to be determined by further anatomical examinations.

Shortly after this, Iwanoff published the results of a case the microscopical details of which were as interesting and have remained as permanent to this day as have the ophthalmoscopic appearances first detailed by Nagel.

Iwanoff's investigations were made upon an eye which had been penetrated by a foreign body, and which had been enucleated by Wecker on account of sympathetic trouble in the fellow-eye.

On opening the eye, the papilla appeared to be much swollen. The retinal vessels from the entrance of the nerve up to the ora serrata showed themselves in the form of white tubes, which contrasted slightly with the background of the retina, which was somewhat disturbed. These alterations extended as well to the smaller as the larger vessels, and were present upon the venous as well as the arterial branches. Under inspection with a magnifying-glass it was observed that the changes, while still persisting, became gradually less in degree toward the periphery, and that the greater vessels in the neighborhood of the nerve were not all equally affected. Some of these latter were thickened to double their normal calibre, and appeared as perfectly opaque stripes, which at various parts of the retina protruded to a moderate degree above the level of its surface, while the others were only moderately thickened. The disk was swollen and œdematous. The swelling was not limited strictly to the papilla, but extended somewhat into the surrounding district. The increased thickness of the papilla was caused chiefly by an hypertrophy of the connective tissue of the stroma and the vessels, which had become twice as thick as normal. The œdema was due to an exudation into its parenchyma.

The microscopical examination showed that the entire connective tissue of the stroma of the retina was thickened. All the retinal elements were separated from each other, and were surrounded by a parenchymatous exudation, the appearance of which, as a rule, denotes the earliest stages of an acute inflammation of the retina. The changes in the vessels were out of proportion to the moderate degree

of trouble in the nervous and connective-tissue elements. The lumen of the vessel was not encroached upon, was indeed entirely unchanged, and was well filled with blood-corpuscles in a good state of preservation. The endothelium of the inner surface could hardly be called strictly normal. The middle layer (and this is a most important point) appeared perfectly normal. The entire pathological process was concentrated chiefly upon the outer covering of the vessel. The connective tissue of the adventitia was converted into a dense mass of nuclei, which were surrounded by an extremely thin layer of protoplasm, and which lay in a network of excessively delicate fibres.

Upon the nerve these nuclei surrounded the vessels upon all sides in series which varied from ten to twelve deep. The difference between the behavior of the arteries and veins in this region could be readily seen; for, whereas the mass surrounding the arteries consisted of five or six layers of nuclei, that around the veins was made up of only two or three layers. This distinction could not be followed out

FIG. 6.

in the retina proper so accurately as upon the disk, as in the retina some arteries were found which were nearly normal, while, on the other hand, there were veins which were surrounded by ten or

twelve layers of nuclei. The capillary vessels also participated in the trouble, though not to so marked a degree. There was evident proliferation, however, as there was an increased quantity of nuclei. Still, the proliferation here never amounted sufficiently to form an unbroken layer round the capillary vessel. Jaeger, however, furnishes a picture (Fig. 6) in which a fine network of white lines was seen in different parts of the retina with the ophthalmoscope, and which Mauthner, who also saw the case, thought must be due to a hyperplasia of the adventitia upon the ramifications of vessels which were of too small a calibre to be visible under normal conditions.

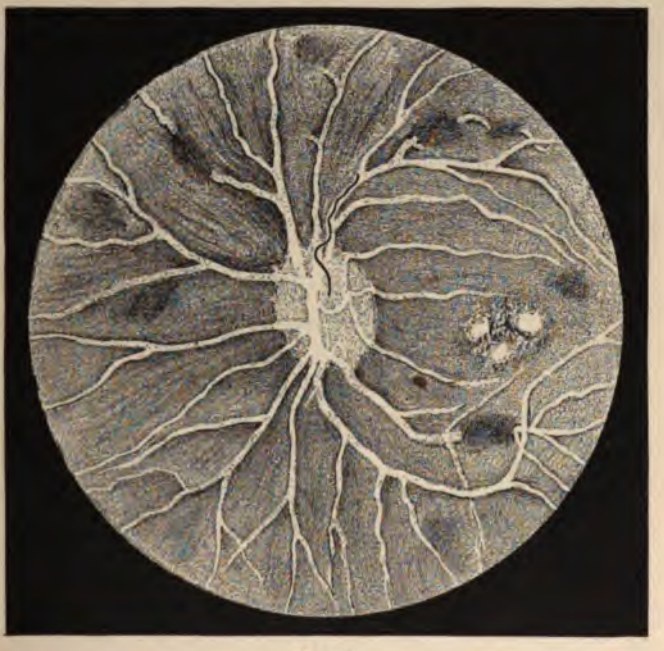

FIG. 7.

Fig. 7 is from an article on thrombosis and perivasculitis, by Dr. George C. Harlan, who gives the following description of the ophthalmoscopic appearances:

"There was no light-perception in the eye. The media were clear. The ophthalmoscope showed hæmorrhages scattered all over the retina; none of them, with two or three exceptions, at all dense. Some were striated, and extended in long streaks by the sides of the vessels, were found at all the bifurcations, and in a few spots concealed the vessels. Others were in large, rounded, faint patches, with difficulty distinguished from the surrounding fundus. At the

macula there were three groups of white spots imbedded in dark pigment. The disk was opaque-looking and snow-white, its margins slightly blurred. One thread-like arteriole, arising independently near the centre of the disk, and passing upward about two disk diameters, contained red blood. With this exception, all the retinal vessels, both arteries and veins, were converted into white bands and lines, appearing as if marked out with chalk."

FIG. 8.

Fig. 8, given also by Dr. Harlan by way of comparison, "is that of a negro woman, beyond middle age, partially blind for a long time, with no definite history, but with evident marks of extensive intraocular inflammation. The right eye was quite blind, with wide and firm posterior synechiæ of the iris and dense inflammatory cataract. In the left eye, V. $= \frac{20}{100}$. There were several slight synechiæ, the lens was hazy, obscuring but not concealing the fundus, the disk was pale, and there was a pigmented choroidal atrophy at the macula. A large artery running almost directly upward was completely converted into a white, chalky-looking band, with somewhat ragged margins and of varying width. There was no red blood seen in it from its entrance on the disk to the farthest point to which it could be followed. In two smaller arteries passing down-

ward the red color was replaced in parts by irregular segments of the same white appearance; and there were some smaller patches on one of the large veins. On this dead-white ground were scattered numerous scintillating specks, which looked like crystals of cholesterine." *

Inasmuch as the process concentrated itself almost entirely upon the vessels and the tissue immediately surrounding them, and consisted of an enormous proliferation of the nuclei of the adventitia, Iwanoff proposed the name of perivasculitis, and by that name it has been designated ever since. When the trouble is either confined to or predominates in the arteries, a further distinction is sometimes made by some authors, who then speak of periarteritis. Under the title of general periarteritis in a case of chronic renal disease, Dr. Mules reports and gives a drawing of a case in which the branches of the arterial tree over the whole area of the retina had nearly twice their normal width, and resembled a series of snow-white cylinders, unrelieved by any red color, except that here and there in the secondary and tertiary divisions a slight interruption in the opaque sheath allowed dots of red to be seen, showing that the blood-current state persisted.†

Wedl has also described, in his "Atlas," a form of perivasculitis in the principal branches of the arteria centralis. In this case, just as in that described by Iwanoff, the lumen of the vessel was not encroached upon. The mass consisted of very fine nuclei, with the brilliancy of fat, and was looked upon by Wedl as an atheromatous degeneration of the arteries. ‡

I am not aware that atheromatous degeneration has ever been as yet diagnosticated with the mirror.

Chalky Infiltration of the Walls of the Vessels.—This may reach to such a degree that sometimes in chronic inflammations the entire wall presents the appearance, under the microscope, of rigid, white, opaque cylinders. Fatty degeneration and calcification of the walls of a pronounced degree must therefore be accompanied by sufficient opacity to render them visible with the mirror so that they could be seen in such conditions as renal and other forms of retinitis, especially those of a chronic type. But I am not aware, however, that the white appearances which would naturally be produced by calcareous deposits have even been certainly differentiated from the similar appearances produced by vasculitis or some of the forms of fatty degeneration and sclerosis.

* "Trans. American Ophth. Soc.," 1886, p. 199.

† "Trans. Ophth. Soc., United Kingdom," vol. ii., p. 47.

‡ "Atlas der Histologie Ret. Opticus," 1–5.

In all these forms of sclerosis or perivasculitis when a vessel presents a whitened surface which extends across its entire diameter it is sometimes extremely difficult to tell whether the lumen is encroached upon or not. If the changes are limited to one part of the vessel, so that the red current can be seen both above and below the whitened space, then the vessel is patulous, and that it is can be further proved by the fact that the vessel becomes blanched under pressure, and that the red column returns both above and below as soon as the pressure is removed. I am particular in mentioning this, as I can not help thinking that the masking of a vessel by surrounding tissue, especially when the part of the vessel thus concealed is of small extent, has led to the erroneous conclusion that the calibre of the vessel was reduced, or that it was completely closed. A further means of ascertaining whether the current was passing through the vessel has been proposed by Nagel, and consists of throwing the image of the flame not directly upon the crown of the vessel, but a little to one side. The light in this way is reflected somewhat behind the vessel, which the observer then sees by transmitted light, and if the opacity is not too dense, the red color of the blood-column is seen through it.*

To this method may be added that proposed by Liebreich. If the vessel is not changed for its entire length, the observation is to be fastened upon a part which is still red, and the contours of the vessel followed along from this part. If the vessel is not carrying blood, the borders of the white portions are continuous or flush with that of the red. If, however, the white, band-like formation is due to an increase of connective tissue of the adventitia, the whitened portion of the vessel then appears thicker than the red, its borders less well-defined, and more feathery in appearance.

To recapitulate briefly, we see that inflammation of the walls may occur under three principal forms: Firstly, a transparent infiltration, which, in spite of its transparency must be looked upon as a sclerosis, as it contains a large quantity of connective tissue. Secondly, a non-transparent form, in which the entire thickness of the wall is affected, so that the vessel gives the appearance of a white cord when viewed with the mirror, but one in which the outside dimensions of the vessel are not much if at all greater than normal, but which upon section may show, as does the transparent variety, a great reduction in its lumen or a total occlusion. Thirdly, a form where the process is concentrated chiefly upon the outer envelope of the vessel and the surrounding tissue. In this form the vessels appear to be much enlarged externally, while the lumen is very little or not at all affected.

* Zehender, 1864, p. 400.

These morbid processes may for convenience be designated under the general heading of vasculitis, with a further discrimination into arteritis and periarteritis, phlebitis and periphlebitis.

The more extensive forms of the trouble, in which all or the greater part of the vessels of both systems are affected, are comparatively rare. Modified degrees of the same trouble—that is, where a few of the vessels or a single vessel or parts of vessels are affected—are exceedingly common, and are found, or may be found, in every form of inflammation which attacks the retina, either as a consequence of such inflammation, or as a primary affection of the substance of the vessel itself.

Vessels of New Formation.—Strictly speaking, vessels of new formation are very rare, either upon the surface or within the substance of the retina itself. That we often see an increased vascularity in the retina under disturbed conditions, especially upon and about the disk, is undoubtedly true, but it is extremely difficult to tell whether this is due to an actual formation of new vessels or simply caused by the distention and extension of vessels which had already existed, but which, before the disturbance of the part, were too small to be visible with the mirror. The evidence that it is due to the latter and not the former is very strong: firstly, because the so-called vessels of new formation run in the same direction and have the same general plan as the original vessels, and very seldom, if ever, run toward a common centre or focus, except in the case of new growths, when they form a very important diagnostic mark; secondly, we very rarely, if ever, see these vessels of new formation in those cases in which it would naturally be supposed they would be most likely to occur—that is to say, in the effort to establish a collateral circulation. Thus, in embolism or thrombosis, no one has yet seen a well-established anastomosis of vessels, or any undoubted evidence of even a collateral circulation, either carried on by newly-formed vessels or by extension of those which pre-existed. I do not mean to assert by this that vessels of strictly new formation do not occur, but only to point out the exceeding rarity of their occurrence in comparison with increased visibility of vessels which already exist, but which are too minute to be seen with the mirror. That these newly-formed vessels occur in localized and circumscribed spots is also true. Thus Nagel, in the case already referred to of perivasculitis, mentions the fact that a congeries of very minute vessels existed upon the disk of one eye, which were so minute and so thickly congregated together as not to be recognized as individual vessels, while in the other eye, where the process had not advanced so far, individual loops could be discerned. I have myself

seen precisely the same thing in a case of neuro-retinitis of a low form, where, upon the outer portion of the disk, there was a round, ill-defined reddish spot, which was shown to consist of a collection of minute vessels, from the fact that, under a very moderate pressure, the spot became blanched, and regained its rosy-red color as soon as the pressure was relaxed.

Vessels of new formation may also occur in some forms of retinitis, and are then usually distinguished by two characteristics: firstly, that the vessels are usually very fine and carry the same diameter for long distances, with little tendency toward tapering; and, secondly, that they almost always show a marked tortuosity, often passing into corkscrew convolutions. In a case of retinitis in a young man, which was under observation for over two years, and which was accompanied by repeated—almost innumerable—hæmorrhages into the anterior parts of the vitreous, there was a most remarkable development of newly-formed vessels, which presented both of the characteristics mentioned above. From one of the temporal branches of the inferior vein a vessel ran for quite a distance, doubling and twisting upon itself in small spiral convolutions, until it finally arched around the macula lutea. This vessel was met by another of precisely the same character, which proceeded from the upper temporal vein, and which arched downward until the two vessels seemed to join each other in a series of minute corkscrew twists, beyond and a little above the yellow spot. Finally, newly-formed arterial twigs, of the same tortuous character, developed themselves and gradually extended in length. They were so fine, and of so similar an appearance with the veins, as only to be distinguished from them with great difficulty.

Sometimes in glaucomatous excavations of the nerve we meet with vessels of new formation, which may be so fine and occupy so much of the floor of the excavation as to give the impression that a hæmorrhage has occurred. I have had for a long time under observation, a case in which the convolutions of the vessels were so fine, and so closely packed together, that I should certainly have believed the appearance to be due to hæmorrhage, if it had not been for the fact that, in the other eye, in which the process was not so highly developed, the separate twigs of the vessels could be detected. Moreover, the spot became blanched upon pressure.

Another curious case also occurred in my practice, which at first I was inclined to look upon as isolated aneurisms occurring in different parts of the fundus and upon different vessels. The patient at the time was suffering from diabetes, and both eyes exhibited signs of a mild form of retinitis. This was more marked in the left eye than in

the right, and there were a few spots of fatty degeneration near the macula. But the remarkable point in view was the peculiar appearance of the smaller arteries and veins, upon which were clustered isolated and nearly perfectly round formations, looking very much like currants upon a stem. They were very regular in shape.

It was hard to decide what the peculiar bodies were, and hæmorrhages certainly suggested themselves, but this idea was given up, because the formations were much too regular in shape and boundary, and were of a more delicate and transparent character, with a livelier color; and, moreover, they did not alter materially in shape or color for the space of over two months during which time the patient was under observation. I finally came to the conclusion that these circumscribed red spots were not aneurisms or ecchymoses, but were due to congeries of very minute vessels which were too small and too closely woven together to be seen individually as vessels, under the moderate enlargement furnished by the mirror. I was further strengthened in this opinion by calling to mind that Von Graefe had many years ago reported a case where on section of the eye he had discovered some round red spots in the retina, which in the beginning he had taken for ecchymoses, but which later proved to be made up of loops of vessels of the most minute description. With a simple magnifier they appeared like little clumps of red mold upon the surface of the retina; but under a somewhat increased magnifying power (twenty diameters) they were seen to be composed of convolutions of infinitely fine vessels, which projected to a slight degree above the level of the retina. They were distinguished from ecchymoses by their more delicate color and their more circular form. Graefe looked upon their presence as pointing toward the formation of a collateral circulation. Fig. 9 represents the appearances as given by Graefe, under an enlargement of twenty diameters.* When I saw my patient a year after this, these round, cherry-like spots had entirely disappeared. I have been particular to go into this detail concerning these spots because I have at various times seen similar formations in single examples, but never to the same degree.

FIG. 9.

Vessels of new formation are very common in morbid growths which occupy the tissue of the retina, or which protrude from it into

* "Archiv für Ophth.," Band i., Ab. I., p. 381.

the vitreous. They are often of great diagnostic value, and will be dwelt upon further under the heading of morbid growths.

Anastomosis of the retinal vessels, either with each other or with those of the choroid, is also very rare, and the following case, represented by Fig. 10, is inserted here because it is the most perfect and

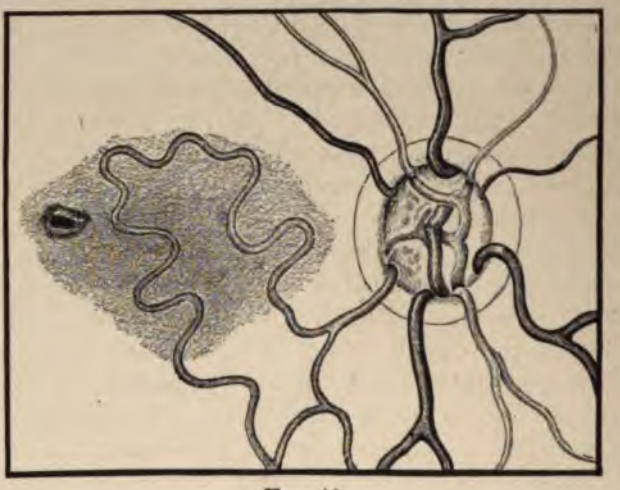

FIG. 10.

the most evident example of both the above conditions which I have yet seen. It occurred in the eye of a middle-aged woman who had lost the left eye in early childhood, which had atrophied to a mere button. Until about four months before visiting me she had had no trouble from her right eye, when she was attacked with all the rational signs of acute glaucoma, which passed after a time into a chronic condition, from which she was still suffering, though in a reduced degree, at the time of her visit.

As the patient was suffering a great deal of pain, and as the nature of the disease was very apparent, although no view of the fundus could be obtained on account of the troubled condition of the media, iridectomy of the right eye, with removal of the atrophied bulb of the left, which was also inflamed, was performed at once. The vision in the right eye was so bad before the operation that only an intensely bright light could be perceived. After the operation, however, vision finally improved so much that the patient could go abroad, and count fingers, in a good light, at ten feet.

The media cleared up, so that a perfectly good view of the fundus could be obtained; and the figure represents the ophthalmoscopic appearances three months after the operation.

As will be seen, the nerve was deeply excavated, and presented in all respects the classic picture of glaucoma. A deep-gray opacity in the retina extended from the outer edge of the optic nerve to the macula, which appeared as a blackish oval spot. Through this gray opacity, and occupying a part of the retina usually free from vessels, ran a branch from one of the principal trunks of the central artery. This vessel was of a considerable size, and pursued an exceedingly

FIG. 11.

tortuous course, twisting in and out in a remarkable manner, till it finally doubled on itself, returning nearly to where it started from, and then passed out of view in the extremity of the field. In about the center of the convoluted part this arterial branch was joined by a short trunk of about an equal or a little larger calibre than its own, which seemed to come up in a sort of rounded, arch-like projection from some vessel lying in the deeper tissues either of the retina or choroid, and by means of which the size of the tortuous vessel was increased, not only at the point of juncture, but also for quite a distance beyond.

Fig. 11 represents not only an anastomosis of the vessels, but what would appear to be a localized collateral circulation. It is the only case of the kind I have ever seen. It occurred in a young man who was suffering from retinitis syphilitica. (See Plate I., Fig. 2.)

There was an extensive hæmorrhage, as will be seen from the drawing, which nearly covered the entire disk. In the upper and outer field there was a large, yellowish-white plaque over which ran a mesh of vessels which evidently communicated with each other. There was no elevation of the surface, and I looked upon this plaque as probably the remains of a blood-clot which was in process of vascularization.

Vessels of New Formation in the Vitreous.—These, too, although comparatively rare of themselves, are not of such rare occurrence, it would seem to me, as writers have been in the habit of alleging. When the vitreous humor is clear, these vessels produce a most remarkable and beautiful picture, and thus give the idea, as the retina does not seem to be much if at all involved, that they are independent and primary productions, especially as they extend far into the vitreous, and have no visible or at times only slightly visible attachments and supports in the shape of a delicate web of connective tissue, which is often so thin as to escape detection, except under the closest inspection.

These vascular membranes are evidently of an inflammatory origin, just as the non-vascular ones are, and owe their commencement to some form of retinitis in which the adventitia of the parent vessels or the surrounding connective tissue undergoes a hyperplasia, and then becomes vascularized. Leber has suggested the idea that the point of origin of these membranes may be repeated hæmorrhages, which in time become organized. The inflammatory action which accompanies the formation of these vessels may be of a low or high degree, and, if of the latter, the vitreous in the beginning of the attack is disturbed. The vitreous may subsequently become perfectly clear, and then the vessels are seen in all their sharpness, forming a beautiful and most striking picture, and one which it is almost impossible to represent, even by the aid of the most faithful and skillful delineators. I have seen cases in which the vessels seemed to extend forward toward the observer like branches of coral in a perfectly transparent fluid, with here and there a feathery membrane connecting different branches. In another case the main branches, soon after leaving the retina, divided up into minute smaller twigs, which then looked like the loops of a returning circulation, and gave the appearance of a short, thick, red fringe as it moved to and fro with the varying movements of the eye. The vessels were evidently venous in character.

Coccius mentions a similar case, in which the projecting vessels were connected partly with venous and partly with arterial twigs. Fig. 12 is a drawing from a chromo-lithograph which accompanies the report

FIG. 12.

of a case of syphilitic retinitis by Nettleship, which is briefly as follows: John K——, aged fifty, was admitted at St. Thomas's Hospital, August, 1883. A year previous to this the patient had a chancre, fol-

FIG. 13.

lowed by well-marked secondary symptoms. Vision had been failing for the past seven months or more. In the left eye there were opacities in the vitreous, with an extensive and protruding detachment of the

retina. In the right eye there was a close meshwork of vessels on the outer side of the disk, and another small patch of vessels on the inner side. There were hæmorrhages at the macula lutea and at the periphery of the field. A little later a flat, transparent and vascular membrane made its appearance, attached to the outer border of the optic disk, and projecting straight forward into the vitreous. The vessels upon it were looped and very numerous, one larger one forming its anterior border. The top of the membrane was seen with + 4 D. Six months after, which was when the drawing was made, the vitreous had cleared up, the retinal hæmorrhages had been absorbed, and the condition was that shown in the drawing.* Fig. 13 presents the appearance of the same eye a year and a half later.

Dilatations of the Vessels.—Dilatations or constrictions which are limited to a short district of the vessels—as, for example, varicosities or aneurisms—are much more rare than a general dilatation or narrowing of the entire vascular system. We not infrequently see on the principal vessels, just at the pylorus, a small dilatation, as if the vessel

Fig. 14.

had started with a somewhat larger diameter, which for some reason or other, after a short traject, had undergone a more or less sudden diminution in its diameter, and had then continued on upon a reduced scale, the dilated portion bearing a great resemblance to a cirsoid dila-

* "Trans. Ophth. Soc. United Kingdom," vol. iv., 1884, p. 150.

tation. These may occur in both arteries and veins. Fig. 14 represents such a dilatation on a branch of one of the principal veins. These are undoubtedly at times congenital, but also may be due, at this spot or at more distant districts of the retina, to some inflammatory action. Still, it must be admitted that real varicosities of the vessels are very rare, and have no doubt been confounded by some writers with an abnormally great and corkscrew-like tortuosity of the vessel, in which, however, the lumen itself was not dilated or altered in form.

Nævi.—Schirmer reports a case of great tortuosity of the veins of the retina in a subject who was affected with extensive and deep-seated nævi on various parts of the body, the most interesting of which were those seated upon and within the left eye. Both the upper and lower lids were affected, and the tortuosities of the veins of the retina were so great that they took on not only a lateral but even an antero-posterior curvature. The arteries remained normal, both as to their calibre and course. It was evident that an inflammatory action had existed at some previous time, either intra-uterine or later, as there was atrophy and excavation of the optic nerve, and a redundancy of the connective tissue.*

I have myself seen a case in which the optic nerve was occupied for its lower and outer third by a skein of vessels, which, though much finer than the principal vessels upon the disk, evidently started from them. They formed a very conspicuous network, which was evidently of venous origin, as the vessels were so thickly interwoven as to look like a congeries of red worms.

Liebreich has given a most remarkable drawing of a bead-like series of varicosities of the veins, observed in a case of glaucoma, and which were arranged one after the other, like the pearls of a necklace. This is certainly one of the most remarkable pictures in ophthalmoscopy, and would be pronounced untrue to nature if drawn by a less skillful artist. It would, moreover, appear from the drawing, that the innumerable bead-like sections of the vessel were as much due to constrictions of the vessel-wall as to dilatations. This appearance, which has, to a degree at least, been corroborrated by Pagenstecher, is evidently due to a disease of the walls of the vessel, as simple dilatation from impediment to the circulation could hardly suffice to produce such a result.†

Martin gives a drawing of some isolated varicosities upon the veins.‡

* "Archiv für Ophth.," Band vii., p. 119.

† "Atlas d'Ophthalmoscopie," Tab. XI., Fig. 1.

‡ "Atlas d'Ophthalmoscopie," Tab. VII., Fig. 6.

Aneurisms of the Central Vessels.—Aneurismal distentions of the central artery had already been found on section before the invention of the ophthalmoscope, and such cases had been recorded by Schmiedler, the elder Graefe, Schön, and Scultet; but the first case ever seen with the mirror, of this very rare occurrence, was reported by Sous, some ten years after the invention of the mirror, in the "Annales d'Oculistique" for 1865, Vol. LIII. Upon the papilla of the left eye of a woman of advanced age there was a red ovoid tumor or dilatation, which, when viewed by the inverted image, was seen to pass below the margin of the disk, which then, after becoming abruptly narrower, entered into one of the retinal arteries. The tumor presented evident signs of pulsation, the movements being synchronous with the radial pulse. The arteries were reduced in size, while the veins were somewhat broader, but not tortuous. The vision in the eye was much reduced, and the aneurism was evidently the result of a pathological process, and not a congenital defect.

Fig. 15.

Dr. Francis B. Loring has reported an interesting distention of the walls of the lower branch of the central artery, which is particularly well marked and distinct in all its details. This is shown in Fig. 15, and was discovered in the right eye of a deaf-mute, who was

suffering from a slight and temporary attack of phlyctenular conjunctivitis.

"Both eyes were emmetropic in refraction; V.=$\frac{20}{20}$−, A. good, and the field and color-perception normal. Under the opththalmoscope the media and lenses were clear and transparent, and the retina and choroid normal in every respect. In the left eye, however, the lower branch of the central artery presented a most peculiar bulging of its entire circumference. This annular enlargement took place below the margin of the papilla, at a distance corresponding to about three fourths of its diameter, and was situated at about two thirds of the distance between the lower edge of the disk and the first bifurcation of the artery taking place below it, its central diameter being nearly three times as great as that of the vessel proper. Its color was lighter than that of the artery, and the "light-streak" terminated abruptly at its origin, beginning again immediately below it. There was no pulsation attainable by pressure, and there was no stasis of the blood-column above it. The fundus was singularly free from blood-vessels in its near neighborhood, thus allowing a most exact examination to be made with ease. The patient was a young and healthy man of twenty-five years, and a careful examination of heart and lungs proved both to be normal. His hearing had been lost in early infancy, from an attack of measles." *

Under the title of "Aneurisms on retinal vessels in a peculiar case of retinitis," Mr. Story and Mr. Benson have published a singular case of vascular dilatation, the chief features of which were as follows: The ophthalmoscope showed that the vessels in the outer periphery of the fundus of the right eye were dilated in places. Three bright-red, well-defined, and globular protrusions, which resembled cherries, were observed upon one of the walls of the artery which ran above the macula lutea. There was, moreover, a cylindrical dilatation on the trunk of this artery, which became enlarged so suddenly and evenly as to have quite the appearance of an intussusception. The appearances described are shown in Fig. 16. A question might arise, in regard to the small and perfectly circular spots, as to whether they were not due to congeries of very minute vessels, such as have been found by Graefe anatomically, and supposed to have been seen by myself, with the mirror. This case was again presented to the society by Mr. Story, in 1885, or over ten years later. There were still many aneurismal dilatations of the retinal artery and veins.

Gowers also gives a case of retinal changes from chronic renal disease, in which, besides signs of extensive perivasculitis, there were

* "Trans. American Ophth. Soc.," 1880, p. 40.

also dilatations of the vessels. The arteries, except for the changes due to perivasculitis, were, for the most part, normal. One of these, however, which passed directly downward, presented, at some dis-

FIG. 16.

tance from the disk, at least four distinct dilatations, which, according to Gowers's belief, were evidently minute aneurisms.*

Mannhardt † reports a case of aneurism of the central artery in the region of the disk, the result of a traumatism, which was accompanied by rupture of the choroid. In this connection, it is very curious that, where blows upon and around the eyes are so numerous, and where direct traumatism from penetration of foreign bodies is so common, cases in which there is a direct connection between arteries and veins should be so very rarely observed. I have never seen a well-authenticated case, although I did once observe one in which it was supposed, not only by myself but by others, that such a connection might be present. The lower principal artery and

* "Medical Ophthalmoscopy," p. 366, Plate XII., Fig. 1.

† Zehender, vol. xiii., p. 133.

vein were in the closest contact possible, but there was no actual communication between the currents of the two vessels, and there was no change in the bright color of the arterial blood, either at the supposed place of puncture or below it. There were, however, evidences of past inflammatory action in the great tortuosity of the vessels, and in the broad white bands which accompanied both the vein and artery for a considerable distance.

Magnus,* however, reports an exceedingly interesting case, and one which would appear to have been a true aneurismal varix of trau-

FIG. 17.

matic origin, in a hospital nurse who, two years before, had received a severe blow upon the affected eye. This was considerably injured at the time and much swollen. The vision, which was perfectly good before the blow, was so impaired at the time of the examination that

* Virchow's "Archives," Band lx., page 38, 1874.

the patient could with difficulty count fingers. The mirror showed that all the vessels which left the papilla were three or four times their normal calibre, and presented a uniform deep-brownish-red color. This was so marked that it was impossible to differentiate the arteries from the veins, except in the most peripheric branches. Fig. 17, which is drawn with the inverted image, represents the ophthalmoscopic appearances. From the lower and right-hand quadrant of the papilla two vessels emerge, which run side by side for a short distance, and pursue a very tortuous course over the retina, in order to come together again at a distance of about two or three diameters of the disk. It could be determined, by following along the vessels for a considerable distance, that that toward the nasal region was the artery, and that toward the temporal region the vein. There is also in the upper and left-hand quadrant of the figure very nearly the same conditions. A vessel which runs from the disk toward the nasal side of the field (that is, toward the left of the drawing) passes another vessel which comes from the upper and inner part of the disk, and the two, after forming a convolution, enter into direct communication with each other. Magnus is of the opinion that these appearances, when taken in connection with the traumatism, are due to an extensive laceration of the vessels and the retina, accompanied by hæmorrhage, and that a subsequent connection of the two vessels followed, which formed an arterio-venous union, the diagnosis being confirmed by the peculiar appearance of the blood in both sets of vessels.

Arterial Pulsation.—It has already been pointed out that an arterial pulse may exist in a normal eye; and, although its occurrence is to the ordinary observer extremely rare, I am informed by my friend Dr. Wadsworth, who has made quite a study of the matter, that by careful scrutiny it can often be detected, and must, therefore, have been often overlooked from want of care.

When a spontaneous arterial pulse is present in pathological conditions, it is usually due to two principal causes, intraocular pressure and organic disease of the heart, in each of which it manifests itself by peculiarities which it is important to notice.

When due to intraocular pressure, or, more accurately put, wherever the pressure on the outside of the vessel is from any cause greater than that within, the pulse then manifests itself by alternate contractions and expansions of the blood-column, which are sometimes so rapid and so vivid in character as to be almost luminous—especially, as is not infrequently the case, when the vessels upon the disk completely empty themselves.

This peculiar open-and-shut movement is almost invariably con-

fined to the region of the disk, and rarely, if ever, extends beyond the limits of the papilla, thus radically differing from the pulsation due to heart-disease, which not only extends beyond the borders of the disk to a greater or less degree into the retina, but which at times may be visible in the smallest vessels, even to the very periphery of the field. The distinction above alluded to is not, however, invariable; for, curiously enough, Jaeger, who first discovered the existence of a spontaneous arterial pulse, did so in a case of glaucoma, in which not only were the pulsations present upon the disk, but where they could also be followed far into the retina, and these pulsations were quite of the character of those later described by Quincke and Becker. A question arises whether in this case there was not a combination of the two diseases, glaucoma and insufficiency of the valves. The question is all the more allowable, as I myself have seen a case of marked glaucomatous excavation of the optic nerve in which the pulsations extended into the retina, and where, upon examination, insufficiency was suspected. The patient was sent to a more expert colleague for an opinion, and unfortunately never returned.

The arterial pulse, when due to pressure, occurs usually upon one branch of the artery, but sometimes does so on several, or, indeed, upon all the branches situated upon the disk. The distention of the artery occupies a longer space of time than the contraction—that is to say, a gradual filling up takes place, followed by a momentary rest, which is followed in turn by a sudden emptying of the vessel, which consumes only about half the time that the influx occupies. The pulse upon the disk is synchronous with that at the wrist.

The arterial pulsations seen in diseases of the heart may be divided into two classes—an arterial and capillary pulse. Both of these varieties of pulsation, as incident to heart-disease, were discovered by Quincke, who observed that, in certain cases of insufficiency of the aortic valves, besides a strongly-marked pulsation of the arteries which extended far into the retina, there was also a regular and rhythmical flushing and paling of the disk. The increased redness was synchronous with the systole, and the bleaching with the diastole of the heart, and could be accounted for only by the alternating increase and decrease in the quantity of the blood in the smaller arterioles and capillaries.

The alternations in color were most conspicuous in the middle of the disk, and especially upon the borders of the physiological excavation. They could not be traced beyond the borders of the papilla, even if they existed, on account of the lack of contrast in the red background of the choroid.

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The appearance of this form of pulsation occurs only in the more marked cases of aortic insufficiency, and even where it does occur it is not at all times present.* When a venous pulse is present in these cases, it is usually increased in character.

The existence of this capillary pulse was corroborated by Schmidt; but Becker, who has made an exhaustive study of all the pulsations to be observed in troubles of the heart, declares that he has been unable ever to see this systolic change in color, described by Quincke, as occurring in aortic insufficiency. It is interesting in this connection to remember that, according to the experiments of many observers, there is no flushing of the disk from the use of nitrite of amyl, even where the face and ears have been very much suffused. I have never seen this form of pulsation myself, but I do not wish to imply from this that it does not ever occur, but merely to emphasize its rarity. Dr. Polk has demonstrated the existence of a capillary pulse in the normal eye, and there is no reason why this should not be increased by disease, and manifest itself upon the optic disk, as described by Quincke.

The second form of pulsation which has been so thoroughly dilated upon by Becker, and which relates to the larger vessels, is much more common than that which has just been described as pertaining to the smaller vessels and capillaries. The two most important characteristics of the pulsation are, first, a dilatation of the wall of the vessel; and, secondly, a movement or displacement of the vessel itself. It has but little similarity with the arterial pulse which occurs in glaucoma; for, while this is only an intermitting flow of arterial blood into the eyeball, in which the calibre of the vessel itself is not affected, the arterial pulse due to insufficiency of the valves shows all the characters of a true pulse, such as can be discriminated by the finger at the wrist. Thus, the frequency and the force of the action of the heart can be watched by the eye, and the distention and locomotion of the vessel be noted, and the precise share of time which is occupied by the distention, pause, and contraction of the artery, can be determined, while the true character of the pulse-wave in all the gradations of its curve can be estimated.

These pulsatory phenomena vary in distinctness in various places. The distention of the artery is most clearly seen at the point of division of one of its branches; the locomotion or displacement of the vessel, on the other hand, where an S-shaped curvature occurs in the course of the vessel. During the diastole of the arteries the curvature formed by this S-shaped portion becomes more marked, and then returns to its original dimensions, when the systole takes place.

* Dr. H. Quincke, "Berliner klinische Wochenschrift," 1868.

Thus, in many cases, if such an expression may be used, the entire retina, in those cases where the pulsations are marked in degree, seems to take on an animated expression. Moreover, when a suitable arrangement of the vessels upon the disk is present, the entire stem of the artery may be seen, at every contraction of the heart, to start forward into the vitreous; so that here, too, a peculiar rhythmical movement of the artery is observed. From an exhaustive study of many cases, the conclusion was arrived at by Becker that a true arterial pulsation occurred in almost every case of insufficiency of the aortic valves, which was not complicated with other valvular lesions, and that it was the more pronounced the greater the hypertrophy of the left ventricle. It was only in those cases where, with the insufficiency, there was also in combination anæmia, or fatty degeneration of the heart, that the pulse was not visible. In many cases there was also a well-marked venous pulse, not only of the kind which is ordinarily seen, and which consists of rhythmical interruptions to the outflow of the blood at the porus opticus, but also a true pulse, in which a change in the calibre of the vessel occurred in the retina before the vessel had arrived at the disk.

That arterial pulsation, under the above conditions, is a pretty constant accompaniment of insufficiency of the aortic valves, is no doubt true, but, that it is so invariably the case as Becker would seem to imply, would appear to be doubtful, as I have myself seen cases of marked aortic insufficiency without complications, where there was no sign that I could detect of any arterial pulsation.

A rhythmical pulsation of the arteries, such as has just been described, occurs also in aneurism of the aorta.

Becker also calls attention to the fact that, inasmuch as these pulsations take place in eyes in which there is no increased intraocular pressure, and indeed in perfectly normal eyes, too much stress should not be laid upon their presence, either as a sign of intraocular pressure, or, what is more important, as a prodrome of glaucoma—a sign which has been dwelt upon with so much emphasis by Von Graefe * and others. While this last statement is true to a certain extent, it is also true that spontaneous arterial pulsation is so rare in normal eyes, that when it does occur a careful examination should be made as to the condition of the vision, and especially as to the extent of the field; since the presence of the pulse and the comparative ease with which it can be produced are such valuable symptoms of glaucomatous conditions, that they should never be lightly ignored. Indeed, arterial pulsation is often the deciding straw in making a differ-

* "Archiv für Ophth.," Band xviii., p. 206; Zehender, 1871, p. 380.

ential diagnosis between glaucoma simplex, of an atrophic nature, and excavation of the optic nerve, in which pressure is exerting a deleterious effect, which it may need an operation to counteract.

In this connection it must be borne in mind that a pulsation may occur in the arteries from some compression which has its seat either in the orbit or in some portion of the stem of the nerve, before it reaches the interior of the eye—as, for example, from some morbid growth, or from some peculiar twist of the vessel; and that this may happen in eyes in which the pressure is either normal or less than normal. Thus, I have seen a case of pulsation in the lower branch of the central artery in an eye, which was perfectly normal, in which there was no excavation, and where, too, there was no sign of any trouble of the heart—a pulsation which was so great that the artery emptied itself with each contraction of the heart. It was confined to the limits of the papilla, and was evidently due to some extraocular impediment to the circulation.

Mauthner also makes the general assertion that pulsations of the central artery of the retina may be perceived when the action of the heart is reduced—that is to say, when the number of contractions is reduced, or these are too weak to throw a continuous stream of blood into those parts of the arterial system upon which a certain amount of external pressure is exerted.*

Visible Circulation in the Retinal Vessels.—Jaeger many years ago discovered a peculiar form of circulation in the retina, which, though since seen by a considerable number of observers, is still so rare as to form one of the greatest curiosities yet revealed by the mirror. This consists in the ocular demonstration of the passage of the blood in its course through the retinal vessels.

In the cases described by Jaeger there is at the outset a gradual distention of all the vessels, venous as well as arterial, which is disproportionately large in the arteries, until these become as broad in diameter and as dark in color as the veins. This may be carried to such an extent that the arteries may become three times and the veins twice as broad as in the normal condition, and this increase in diameter is disproportionately greater in the smaller vessels. An actual increase in length or tortuosity does not usually occur. The light-streak is diminished in brilliancy, and at the time of the most marked congestion is entirely absent. With these symptoms there is also some loss of brilliancy in the fundus, and some increased redness of the disk and surrounding retina. The color of the blood is usually the same in both arteries and veins. It is of a strictly venous charac-

* "Lehrbuch der Ophth.," p. 335.

ter, and of a dark and reddish-blue tinge. The most interesting appearance in these cases is the visible retardation of the blood-current. In both the veins and arteries a peculiar movement is visible, as if fine sand were passing with great rapidity through a glass tube. This appearance, at first indistinct, gradually becomes more and more apparent; the movement decreases in rapidity, and the column of blood assumes more and more a granular character, until little by little the conviction is created that one can recognize with certainty that dark-red granular bodies are suspended in a colorless, transparent fluid. These move with considerable rapidity, which in the arteries is in a centrifugal and in the veins a centripetal direction. These red, granular masses gradually increase in size, the interspaces between them also becoming greater. When these granular masses have so increased in size that the diameter corresponds to the lumen of the vessel, they then gradually also increase in length. As the retardation in the movement of the blood becomes more marked, it becomes distinctly seen that the blood has separated into two parts, one of which is of a dark-red color, the other transparent and nearly colorless, with, however, a delicate, reddish-yellow tinge—that is to say, the blood-column is divided into alternate sections of red and colorless fluid. These cylindrical sections correspond in breadth to the lumen of the vessel, while their length increases according to the diameter of the vessel in which they are situated—the smaller this is, the longer the cylinder. Thus, in the principal branches the length of the cylinder is twice or three times its breadth; in the medium-sized vessels it is from four to eight times its breadth, while in the smallest vessels for the most part the cylinders are so long that they occupy the entire length of the vessel, even while the current is passing for a considerable space of time. These cylinders are of different lengths, even in the same vessel, and the length changes frequently either by coalition or separation—a shorter cylinder joining with a longer one, or a longer dividing into shorter ones. The more transparent portions occupy the spaces between the red portions, and like them fill the lumen of the vessel and form sections of various and changing lengths.

When the cylinders arrive in the course of the circulation at a division of the vessel, and the dividing branches are of the same caliber, the cylinders divide themselves pretty equally between the two vessels. When, however, one branch is smaller than the other, or the angle of its departure more obtuse, a smaller proportion of the red and a greater proportion of the colorless sections pass to this branch. When the current in one of the larger arteries meets a considerably smaller branch, the red cylinders are inclined to pass

directly over the mouth of the vessel undivided, or in some cases small detachments from the cylinders pass into it. The calibre of the vessel is always maintained by the colorless portion which seems to pass freely into it. When the branch from the larger vessel is extremely fine, very little, or sometimes none, of the red portion enters it. As the current is more rapid in the smaller vessels, and as these are at times completely filled with red blood, which extends for a considerable distance in length, and as these red portions are alternated by similar sections of transparent fluid, it appears at times to a superficial observer as if the vessels were sometimes filled with red blood, and then again as if they were empty, or as if there was a pulsation in the vessels. This has led several observers to look upon these colorless portions as empty spaces, instead of being filled, as they undoubtedly are, with a transparent fluid.

Very nearly the same phenomena take place in the veins as in the arteries, though there is less uniformity in the various phases, and the sections or cylinders are usually of shorter length. Taken as a whole the rapidity of the current is less in the larger vessels, and increases in proportion to the decrease in the diameter—quicker in the smaller and quickest of all in the most minute. With these variations it is pretty uniform in character and degree, only varying in certain exceptional cases, and in particular vessels, where a retardation or even a complete stoppage may occur for a greater or longer space of time.

When the retardation has reached a certain degree a sudden stoppage of the cylinders can be seen, either in certain parts of the field or over the entire system, which is followed again by another forward movement. After another shorter interval another stoppage occurs, and so on, the intervals between the two becoming gradually shorter and shorter, until finally the mass of blood seems to move forward by successive pushes from behind. After this progressive movement has lasted for some time, and has decreased essentially in rapidity, a sort of to-and-fro movement occurs in the entire mass of the blood, the excursions constantly decreasing in length. This back-and-forth, pendulum-like movement degenerates into a merely vibratory motion of the cylinders, which is then followed by complete stasis. As a usual thing, this first stoppage of the blood is not a lasting one, for, after a period of some five or ten minutes or more, a gradual displacement forward of the red cylinders takes place in the larger arterial branches, for about one half or the whole of their length, while the colorless cylinders become shorter, or fade away, to be reformed in other places.

The stasis, having once taken place, may last for several days.

When it is resolved, it is so, gradually, and pursues in a retrograde way the course it took in its development—first of all a quivering motion, then a to-and-fro movement, then an interrupted, pulsatory progression of the cylinders, first in particular regions, and then in the entire field; and then, out of this interrupted movement, little by little, a uniform and continuous flow is developed, with a continually increasing rapidity, until it is difficult to recognize the single cylinders, and until, finally, all visible movement is withdrawn from the eye, and the circulation resumes its normal, quiet, and motionless appearance.

There was always a considerable functional disturbance in the cases that were observed by Jaeger, the vision sinking very quickly to quantitative perception of light, and even this was entirely wanting during the stage of complete stasis. With the return of the movement of the blood the sensation of light increased, and vision gradually improved, until it nearly equaled the original amount.

In regard to the nature of this separation of the blood into red and colorless divisions, there can be hardly a question but that the cylinders are composed of aggregated red blood-corpuscles, as Jaeger asserts. It is possible, also, in the light of more recent investigation, that they may be of the nature of aggregated blood-plaques, seen to occur in other organs—at the edges of the openings in ruptured vessels, or where the current is retarded from obstructions or projections into the lumen of the vessel.

Under the influence of the retardation of the current, the central position which the red portion of the blood-column occupies in physiological conditions, and the external position of the plasmic portion, could be much more readily observed than in health.

It is of interest to notice, too, that during the stasis the nourishment of the retina, in the cases reported by Jaeger, was continued, and was sufficient to maintain the integrity of the retinal elements, so that nearly normal vision was restored when the circulation became re-established, and that during this time the change which took place was a decrease in the length of the red cylinders.* This would imply that it was due, in part at least, to the agency of the red aggregations that the nourishment of the retina was maintained. It is also worthy of notice that there was, in these cases, but very little or no œdema, which would go to show that, in certain forms of stasis, where there is a marked diapedesis of white corpuscles, there must be an altered condition of the walls of the vessels which would favor such a transudation.

* "Ergebnisse der Untersuchung," etc., Jaeger, p. 100, *et al.*

Following the discovery by Jaeger of this peculiar form of circulation in the very infancy of ophthalmoscopy, came similar observations by Liebreich, Schneller, Graefe, Schweigger, and lately by a good many others.

In this country, interesting cases have been reported by Dr. Callan and Dr. Schell. Fig. 18 is taken from the case reported by the latter, and shows in a graphic and pictorial way the segmentation of the blood. From the sudden loss of vision and the thinness of the vessels the case was supposed to be one of embolism.*

FIG. 18.

Dr. Callan, on the other hand, was of the opinion that the case examined by him was not one of embolism but of retro-bulbar compression of the optic nerve within its sheath, due to inflammatory products.†

Stated broadly, the appearances noted by these various observers are probably due to some mechanical retardation of the current, and are simply the expression of stasis, whether from overdistention of the walls or lack of propelling force from behind.

I have been led somewhat into detail in regard to this matter, as it seems to bear so strongly on some modern observations on the study of the blood, and because the retinal vessels offer the only place in which these discoveries and deductions can be studied upon the human subject.

Ischæmia of the Retina.—This term has been used to denote a peculiar condition of the retinal circulation, in which the arteries are much reduced in size, sometimes even thread-like, while the veins either remain of their normal dimensions or are somewhat fuller than usual. With this increased fullness there may be slight, or at times a marked tortuosity. On the other hand, cases have been reported where the veins as well as the arteries were much reduced in size.

This attenuated condition of the arteries is usually accompanied with total blindness, for the time being at least, of both eyes. As a rule, this reduction in the calibre of the arteries is the only ophthal-

* "Trans. American Ophth. Soc.," 1884, p. 689.

† "Archives of Ophth.," vol. x., No. 2, June, 1881.

moscopic appearance; but occasionally the edges of the nerve are somewhat obscured, the disk pale, and the retina at times slightly lacking in transparency. The attack may last any length of time, from a single moment to a period of several weeks.

The trouble has been supposed to result from a reduced action of the heart, which, in its weakened condition, is unable to force the blood into the distant parts of the circulation with sufficient force to overcome the intraocular pressure, and in sufficient quantities to maintain the natural functions of the retina. That this is the true cause of the trouble has been shown, it is claimed, by the glittering results which have immediately followed the performance of repeated paracentesis and the use of nitrite of amyl. Heddäus reports a remarkable case of spontaneous cure after a total blindness of three weeks.

Dr. R. H. Derby has reported an interesting case in a young girl, in which there was an extreme contraction of the field of vision, while the central vision remained comparatively good, and was not totally obliterated, as is usually the case. The patient was much benefited by the use of nitrite of amyl. Knapp, Lecondi, and others have reported cases of one-sided ischæmia.

The fact that the blindness only occurs on one side has led to the expression of the opinion, by some writers, that the failure of sight could not be due to reduced energy of the heart, for if it were, the blindness would occur, not in one only, but in both eyes. This does not follow, however, for it may well happen that, from some anatomical peculiarity, less blood may flow into one eye than the other, and that, while the supply is really reduced on both sides, it is only reduced on one side sufficient to interfere with the functions of the retina.

Cramp of the Vessels.—This is a condition which is akin to that just described, if not identical with it so far as the subjective and physical signs are concerned, including the ophthalmoscopic appearances, and it would be impossible, from the statements of the patient or from the appearance of the fundus, to say whether the reduction in the size of the vessels was due to an imperfect supply of blood, caused by some extra-ocular condition, such as a reduced *vis a tergo*, or from a spasmodic condition of the vessels themselves within the eye.

Hughlings Jackson, many years ago, called attention to the contraction of the vessels in a case of epileptiform convulsions which he examined, if not in an actual fit, at least during a convulsion in which consciousness was lost. The optic disks were extremely pale, and once the vessels disappeared altogether for an appreciable space of

time. After a while, however, they reappeared, and were found to vary with the respiration. When the patient inspired, the vessels disappeared, returning again on expiration, like lines of red ink on white paper.* On the other hand, Gowers kept an artery and vein in view throughout the fit, by the direct method of examination, and saw no change in the artery. The vein was distended during the cyanotic stage. Jackson also reports a case of a middle-aged woman who had become suddenly blind, and remained so for fully five minutes. The blindness came on suddenly and left suddenly. It was not total darkness, but "dusk." When Jackson saw her, a minute afterward, she could read well with each eye, and the fundus of each was normal. As Jackson believed that this temporary blindness was due to contraction of the blood-vessels, and was akin to what produced the convulsive attacks in epilepsy, he named this peculiar condition epilepsy of the retina, a name by which the trouble is often designated at the present day.

I have myself seen several of these cases, and have attributed the blindness to a constriction of the vessels, or an imperfect supply of blood, or perhaps a temporary pause in the circulation within the eye. In two cases these attacks of blindness existed for many years, and were sometimes of daily or even hourly occurrence. In one of these cases the attack was at once cut short by the use of nitrite of amyl, and more slowly by the patient—who was a young physician—literally standing upon his head, a mode of treatment which he instituted himself, upon learning that the cause of the trouble might be due to an imperfect blood-supply, and the success of the remedy would go to show that here, at least, it was due to a faulty supply of blood rather than a spasmodic contraction of the vessels.

One of the most remarkable of these interesting cases of cramp of the vessels was that mentioned by Dr. Noyes.† The case is briefly as follows: A mechanic, fifty-five years of age, while talking, gradually began to lose sight. Everything grew dark, and in about ten minutes he was blind and had to be led home. He remained in this state of complete want of perception of light from 2 P. M. until nearly 6 A. M. At 2 P. M., or twenty-four hours after he had lost his sight, he was brought to the infirmary, where Dr. Noyes saw him. He was able to recognize light, and had a dim perception of form. After a careful examination with the ophthalmoscope, it was determined that the trouble was spasm of the arteries, which were nearly empty and were thread-like. There was almost entire suspension of the retinal circu-

* "Ophth. Hospital Rep.," vol. iv., 1863.

† "Trans. American Ophth. Soc.," 1882, p. 370.

lation, $V = \frac{20}{c}$. He was put under the influence of nitrite of amyl, and in five minutes he had completely recovered. $V = \frac{20}{xx}$.

It should be remembered, however, that sudden attacks of blindness occur where there are no ophthalmoscopic signs, or any history of an injury to the head or elsewhere, and where all the bodily functions, except that of sight, remain intact. Thus, I was called once to see a case of sudden blindness in a woman of middle age, who had become totally blind in both eyes only twenty-four hours before. At the time I saw her there was no perception of light in either eye. She had gone to bed seeing perfectly well, and had waked up blind. There were absolutely no ophthalmoscopic appearances, the fundus being perfectly normal in every respect. When I saw the patient three weeks later the fundus was still normal, and there were no signs of commencing atrophy of the disk, as I fully expected would be the case. I did not see the patient again, though I learned from her physician that the conditions remained the same. Had the blindness, in the first instance, been due to epilepsy or cramp of the vessels, the blindness would have passed off with the return of the circulation to the retina, as twenty-four hours was too short a time for the retina to have received a permanent impairment in function. The trouble was evidently of central origin, and that transient attacks of blindness may be due to the same central origin is shown by the fact that, with an absence of all ophthalmoscopic signs, there may be a reduced visual power of a hemiopic character.

Embolism of the Central Artery.—A typical, I had almost said a poetical, case of embolism of the central artery presents a very characteristic set of symptoms, both subjective and objective, which are capable of being summed up in a few words. These are sudden and almost total blindness of a single eye, without pain and without premonition. These subjective symptoms are accompanied by a thread-like attenuation of the arteries, a characteristic circle of œdema occupying the region of the macula, and a still more characteristic cherry-red spot in the centre of this œdematous circle, to which may be added occasional extravasations at different regions of the fundus—preferably, however, near or upon the papilla.

With so definite a cause and with such a distinctive picture it would be supposed that the variations that could possibly occur would be neither great in number nor extensive in degree; and yet so many have been the variations in each and every one of the symptoms related above, in the great and ever-increasing number of recorded cases, that one is almost forced to doubt the accuracy of the diagnosis or the acumen of the observer. Thus the disk may be either

entirely normal or unduly congested or abnormally pale. The contour of the disk may be sharply defined or slightly obscured by a delicate, haze-like œdema, or it may be entirely surrounded by a dark and untransparent exudation. The hæmorrhages which occur may be, as they usually are, small, and occupy, as they commonly do, the surface of the disk itself or its neighborhood, or they may lie between the disk and the macula lutea or in different parts of the fundus. The extravasations are mostly from the veins, though they occasionally also occur from the arteries. They are usually from the larger, and not, as would be expected, from the smaller vessels and capillaries.

But perhaps the most conspicuous variations are those which pertain to the vessels themselves, especially the arteries. These, according to the cases reported, may be either reduced to a mere thread-like current for their entire length, or they may be visible at or near the disk, while their secondary divisions are entirely lost to sight. On the other hand, they may be nearly or absolutely empty, and only to be traced by their walls, and gradually increase in fullness and visibility as they approach the periphery of the field. Or, again, they may be moderately filled at the disk, and more so at the periphery, giving a tapering appearance to the arteries, which is not an infrequent occurrence; or, lastly, they may be narrowed in the middle of the field, while they are moderately or even normally filled at both ends.

When the tissue surrounding the vessel is œdematous and nontransparent, which frequently occurs in the neighborhood of the disk, I am inclined to believe that this reduction in size is rather apparent than real, as I have not infrequently observed in those cases in which the œdema rapidly disappeared, that the arteries at once assumed their normal, or at least their proportionate, size. The contour of the walls, too, instead of being uniform and continuous, is sometimes so changed that the calibre of the vessel varies in different parts, so that a distended or rather a full portion alternates with a constricted or what seems to be an empty section.

The arteries are, moreover, often bordered with delicate white lines, which have sometimes been considered as the visible expression of the walls themselves, which then become thicker and less transparent from contracting upon themselves; they are, however, most probably due to a mild form of perivasculitis. They may be present in the earliest stages of the disease, even when the case is examined almost immediately after the attack, and may go so far as to give the appearance of white stripes inclosing a fine red thread in their centre. Again, in some of the cases that have been reported, and in more

which have been exhibited at societies as examples of embolism, the arteries have failed to show any attenuation at all—have, indeed, been perfectly normal, the sole and only symptom being sudden loss of vision in a single eye.

The veins, like the arteries, may be reduced in size, but are, as a rule, either normal or a little distended. When contracted they are so usually near the disk, increasing in size toward the periphery, sometimes to such a degree as to have a leech-like appearance. They may show the same interruptions and contractions as the arteries, but do so much more rarely. Neither veins nor arteries show much tendency to become tortuous, though cases are reported, especially when the trouble is confined to a branch, where this occurs. Nor is there, so far as I know, ever any increased elevation of the papilla.

The œdema at the macula also varies very much in character and degree. It is usually of a pale grayish, more rarely a bluish, and still more rarely of a greenish tinge. Occasionally it is so dense in character as to have a milky-white or a yellowish-white appearance. It usually occupies the whole of the region of the macula, but does not extend over the fovea, the position of which is marked by the cherry-red spot. Still, a slight veil is sometimes drawn even over this, and Gowers reports a case where the milk-white disturbance totally obscured the red spot. The vessels which are present on the whitened background formed by the œdema are not vessels of new formation, but those that belong to the retina, but which show more distinctly on account of increased dilatation and from contrast.

This œdema may be one of the earliest symptoms, and then, again, not be present until two or three weeks after the attack. It may be present when the vessels are contracted or when they are of normal calibre, or it may be entirely wanting.

Although the milky cloudiness round the region of the macula and the bright, cherry-red spot which marks the position of the fovea are very characteristic of embolism, they are not, as many appear to suppose, absolutely pathognomonic of it or confined exclusively to it. They occur sometimes in specific retinitis, follow injuries from blows, and other causes, and I believe that they may occur whenever the circulation is so much interfered with that stasis results, either partial or complete, in either veins or arteries. In other words, the milky appearance is simply symptomatic of transudation from the vessels into the surrounding tissue, particularly into the inner layers of the retina, and, above all, into the nerve fibre-layer; and the reason why the infiltration is the most dense round the macula is, that it is the place of

all others of the entire retina which is the most supplied with minute vascular twigs (see vol. i, p. 35 *et al*).

From the fact that the nerve fibre-layer does not extend over the region of the macula as a connected layer, and from the fact that most of the other layers of the retina become reduced in thickness or are entirely wanting, there would be at this place a fenestrum or vacant space, through which the underlying choroid would be apparent in case that that part of the retina immediately surrounding the macula should become infiltrated, and the more milky this was the brighter would be the red spot marking the position of the fovea.

In regard to the precise nature of this red spot various views have been maintained. It certainly does not have the appearance of a hæmorrhage. It is generally too uniform in its contour, and remains too long unchanged, and is of too texture-like an appearance to be a simple extravasation; and yet it is of too vivid a color to be merely the mark of the contrast of a normal choroid seen through the retina. It is more probably the combined effect of a congestion of the capillary and minute vascular circulation of the choroid at this place contrasted with an infiltrated retina—a congestion which in some cases may even end in hæmorrhage, but which in all cases may perhaps be looked upon as an attempt to supply through the choroid, nourishment to the retina, the circulation of which has been cut off or interfered with. And this is all the more probable when it is remembered that the capillary circulation at this place in the choroid is the finest and most densely woven of any in the whole body. The abnormal amount of pigment found in this position in sections of the eye, might be explained either as the result of hæmorrhage or congestion, but the ophthalmoscopic appearances do not favor the idea of extravasation into the choroid, while they do strongly that of an active congestion, which is supported by the inflammatory changes that have been found microscopically in this region. That the red spot is in the choroid and not in the retina is shown by the fact that, when an enlarged retinal vessel happens to pass near it, accurate measurements prove, according to Mauthner, that it is in a posterior plane.

The nature and velocity of the blood-current vary in different cases in a marked manner. Thus the circulation may be clearly shown to exist even when it is very much reduced in size, and in these cases it has been assumed that either the stoppage is not a complete one, or that a collateral circulation has taken place anterior to the situation of the embolus. Or the current may be of that peculiar form which has already been dwelt upon, in which there is for a time a current

of alternate red and white cylinders, a vibratory movement which is followed by a complete stasis.

Coagulated blood may occur in shorter or longer sections of the vessels; especially in the finer ones, which run toward the macula.

As a rule there is no pulsation upon pressure in either the arteries or veins, although cases have been reported where it could be produced, or where, as has been alleged, there was spontaneous pulsation in the arteries. I have, however, never seen such myself.

The tension of the eye is sometimes reduced; sometimes, though never markedly, it is increased; or it may be, as is usually the case, perfectly normal.

The progress of the disease is ordinarily as follows: The circulation may, after a longer or shorter period, be in part restored so that the arteries and veins become fuller, or the vessels may still preserve their thread-like character. The walls of the vessels become more pronounced and more clearly visible on account of the hypertrophy of the connective-tissue elements of the adventitia, which is shown by fine white lines along the borders of the arteries and veins, but especially the former. Later on, as the result of secondary retrogressive changes, some of the smaller vessels entirely disappear, while the larger ones do so in part, so that here and there disconnected portions of the blood-column are seen separated from each other by other portions, which are merely traceable as fine red or sometimes as white lines, while at other times there is no visible connecting link, large sections of the greater vessels appearing to be completely absorbed.

The disturbance at the macula lutea fades away sometimes in a few days. It may, however, while it lasts vary in intensity during this period, fading almost entirely away, and then increasing again in extent and density. Later on secondary changes occur here also, which consist of a collection of light-yellow spots due to fatty degeneration. These either entirely disappear or remain in a cluster around the macula, not unlike those which occur in some forms of retinitis. The brilliant little points which are occasionally seen are evidently caused by minute particles of cholesterine.

The red spot at the macula gradually grows lighter as the œdema clears up, until, as a rule, the vivid contrast between it and the rest of the fundus is to a great measure or entirely lost. Occasionally, however, it appears, while it loses its red color, to grow darker in hue, corresponding in appearance to what was shown to be a fact under the microscope in a case examined by Gowers, where the pigment in this region was found to be abnormally increased. The red spot may, on the other hand, continue for a long time after the œdema

has been absorbed, and only disappear very slowly, or it may in rare cases remain indefinitely in a modified form and of a darker hue, when, as is stated above, there is an increase of pigment.

The disturbances in the region of the disk gradually disappear, and the contours of the nerve become more sharply defined. The papilla gradually loses its pink hue, becomes whiter, and finally passes into a state of atrophy which at times resembles simple white atrophy, but which is usually of a more opaque and less brilliant hue, while delicate shreds of connective tissue line the sides or surround the larger vessels, especially at the porus opticus. Oftentimes it is this greater development of connective tissue and the history of sudden blindness in former years that enables the physician to determine that the ophthalmoscopic appearances are probably due to embolism or thrombosis and not to atrophy—a fact which directs his attention for the first time toward the heart, where valvular or other troubles are often detected.

The vision in these cases of embolism, or what have been classed as embolism, is usually, for the time, at least, totally abolished. When the patient has had time and the presence of mind to notice the manner in which the vision has been lost, the impairment of sight has usually been observed to come on with a rapidly concentrating loss of the field until total blindness has been produced. At other times a cloud is said to pass rapidly before one eye, or sometimes, indeed, before both eyes, the obscuration passing from one eye as rapidly as it came, while the other remains permanently blind. On the other hand, the blindness may begin at the center of the field and extend toward the periphery. Central scotoma may also occur at the time of the attack, or, again, an improvement in vision may take place at the periphery, leaving a permanent central scotoma.

The total blindness following the first attack may after a time improve somewhat, and it is then found, as a rule, that the patient has perception of light in the upper and outer portion of the field. The central vision may also improve later to such a degree that useful vision is restored, and cases of complete recovery have been reported. These are, however, exceedingly rare. It has been assumed in these cases that the embolus was either incomplete or had become disintegrated, or that a collateral circulation had taken place, as has been before alluded to, but of which there is as yet no actual proof either ophthalmoscopically or microscopically.

Not infrequently the patient has a sudden temporary loss of sight, which passes off only to be followed by another, which ends in permanent blindness. These temporary attacks of blindness, occurring

at longer or shorter intervals, have been mentioned as taking place in about one fourth of the recorded cases.

Finally, each eye may be successively attacked, making a case of double embolism.

Microscopical Examination.—The number of eyes that have been examined under the microscope which were supposed from the ophthalmoscopic picture to be affected with embolism are very few, indeed, there being only as yet some seven or eight. It is, therefore, very remarkable that in the first case pronounced to be embolism by von Graefe the eye should have come to an examination and that the embolus should have been found. This was the classical case described by Schweigger, in which an embolus was discovered which apparently completely filled the lumen of the artery. This was situated in the region of the lamina cribrosa in the main trunk of the vessel, and had the appearance a year and a half after the attack of a round pellet forced into an elastic tube, the walls of which it still distended.*

The case reported by Gowers is one of the most interesting and one of the most convincing as to the alleged cause since emboli were found in other organs of the body. The case is designated as one of simultaneous embolism of the central artery of the retina and middle cerebral artery, and is briefly as follows:

The patient, a man of thirty, had in early life an attack of low fever, attended by pains, chorea, and weakness in his right arm. A year later he had a similar attack. During the six months before his present illness he suffered from cough, shortness of breath, and occasional slight hæmoptysis. Without premonitory symptoms, he suddenly, when out walking, lost consciousness, was picked up insensible and brought to the hospital. There was complete paralysis of the right arm and leg and slight paralysis of the face, and aphasia. At the heart there was a loud systolic apex murmur and evidences of dilatation of the left ventricle. The urine contained no albumen.

During the following month the patient gradually grew worse. He began to vomit food and cough up blood. There was œdema of the left arm with hæmorrhagic spots up to the bend of the elbow, and signs of effusion of the right pleura showed themselves. Two months after the attack the patient died.

Four days after the attack an examination was made with the mirror, which showed that the right eye was normal, while the left gave all the well-known symptoms of embolism with the exception that there was no central red spot at the macula. The post-mortem examination showed that the right hemisphere was healthy through-

* "Vorlesungen," 1864, p. 138, Taf. III., Fig. 10.

out. In the left hemisphere there were two areas of softening, and the convolutions of the island of Reil were destroyed. There was an embolus in the left middle cerebral artery. The heart was much diseased. The mitral orifice was much thickened and roughened, and was calcareous on its auricular surface. Each kidney contained an infarct, and a similar infarct was found in the spleen.

Microscopical Examination of the Eye.—The central artery in the optic nerve was dilated here and there, but for the most part it was so contracted that its calibre was in most places reduced to a mere line. The walls were thick, but only in proportion to the contraction of the artery. There was no plication of the inner layer. Just behind the division of the artery in the disk there was a dilatation of some size; but in the disk itself the main branches of the artery were reduced to very narrow dimensions, the cavity of one being only $\frac{1}{2000}$ of an inch in diameter.

Here and there, chiefly in the substance of the nerve, but to a less extent also in the disk, minute granular masses could be seen in the artery. The largest of these was situated in the main trunk, in the substance of the optic nerve, about one eighth of an inch behind the lamina cribrosa (Fig. 19). It was an elongated plug $\frac{1}{300}$ of an inch in length and about $\frac{1}{800}$ of an inch in width, coarsely granular in appearance, and contained many dark points. It apparently did not quite fill the vessel, having doubtless shrunk from the sides while the specimen was being hardened. A little distance in front of this was a small spherical mass. The veins in the disk were reduced in bulk, but were much larger than the arteries. In the retina beyond the limits of the disk, about one of its diameters from its edge,

FIG. 19.

numerous dilated capillaries could be seen running inward through the nerve-fibre layer, some vertically, others at an inclination. They appeared as if passing from the choroid to the surface of the retina, but they could not be traced toward the choroid beyond the inner layer of the retina. Near the macula the microscopical examination showed considerable though variable change. In some places all distinctions of layers were lost. The pigment-epithelium was in this part in close contact with and adherent to the external nuclear layer.

It is Gowers's opinion that a collateral circulation must have been established in this case, as the arteries, as seen with the ophthalmoscope, maintained their thread-like dimensions only for a short distance after leaving the disk and then resumed a distinct though narrowed double contour. Moreover, the red spot at the macula lutea, so often described, was absent. This fact is of interest in connection with the differential diagnosis of embolism within the artery from hæmorrhage around it, as absence of it and the presence of early infiltration around the disk are given by Magnus as diagnostic indications of hæmorrhage around the nerve. In the case now described, unquestionably embolic, there was no spot at the macula, and by the fifth day the infiltration around the papilla was very marked.*

It is important to notice here the considerable amount of inflammatory action that was present and the large amount of structural change which was found in the optic nerve, retina, and pigment epithelium.

Mr. Nettleship has also reported a very instructive case of the microscopical examination of an eye that was affected by embolism, in which there were the signs of active inflammation in the nerve, retina, and choroid.†

Partial Embolism of the Central Artery.—Under this title Schnabel and Sachs have recently published six cases of embolism, in which it was found on section in three cases that the lumen of the vessel had been only partially occluded, so that in one case the passage for the blood by the side of the embolus measured one third of the diameter of the artery. In this case a second embolus was found in that part of the lower main branch of the artery which passed over the papilla.

The ophthalmic signs were those in the main that accompany total occlusion of the vessel. The principal difference was that these observers mention the presence of peculiar white spots situated over the vessels in certain parts of the disk or even in the surface of the

* "Medical Ophthalmoscopy," p. 332, *et al.*

† "Ophth. Hospital Rep.," vol. viii., p. 9.

retina at a considerable distance from the nerve. The circumscribed spots had the appearance of scales, and the reflex was wanting where they were present. These spots were not looked upon by the authors of the paper as the embolus itself, but as circumscribed deposits upon or in the vascular walls caused by the presence of the embolus in some manner not yet understood.

Among the nine cases of embolism seen by these observers, there were four which showed that the emboli were incomplete and only partially occluded the lumen of the vessel. In one of the cases each eye of the patient contained an incomplete embolus, which was visible during life, and a third in the trunk of the left arteria centralis retinæ.

The transitory loss of vision which occurs so frequently in embolism before the final and permanent blindness results is explained by Schnabel and Sachs by the fact that at one time these partial emboli close the current either completely or enough to stop the function of the retina, and that subsequently they are displaced to such a degree as again to allow the current to flow by or round them; and when blindness has become persistent it is a sign that they have by another onward movement permanently blocked the current. When, on the other hand, complete vision is again restored, it occurs because the embolus has been removed into some vessel which does not interfere with the function of the retina, or, by the splitting up into finer emboli, which are carried into smaller vessels in the periphery of the field, where they do not obstruct vision sufficiently to be noticed. By following this assumption still further, they imagine that a sort of embolic dust is washed from the soft growths of the valves by the current of blood which, then entering one or both carotids, is finally carried into the central artery, resulting in unilateral or bilateral blindness, which may entirely or partly disappear as soon as the minute particles have been distributed among the smaller branches of the artery.*

Previous to this, Dr. Mittendorf published a case of what appeared to be an embolus visible to the ophthalmoscope. In the upper branch of the central artery, near its first division, an obstruction could be seen that resembled a colorless fibrinous mass. It did not, however, seem to obstruct the artery completely, as a very fine column of blood, resembling a thin red thread, could be seen at the nasal side of the plug. The vessel appeared to be well filled up to this point, but beyond this no circulation could be noticed. On the following day the upper part of the field of vision was decidedly clearer, and the limitation of the field was somewhat less. The apparent embolus had evidently contracted, and had been carried up to a bifurcation of the

* "Archives of Ophthalmology," New York, vol. xiv., p. 263.

main branch, leaving the smaller branch, which it had previously obstructed, free. In this latter position it remained permanently fixed.

The vision of the patient underwent corresponding changes. From the total darkness of the first two days, the upper part of the field became clearer and larger, until the patient regained the vision of $\frac{1}{10}$, and a large part—more than half—of the peripherical vision.*

Embolism of a Branch of the Central Artery.—The closure of a branch of an artery usually only varies from that of the main trunk in degree, as the symptoms are, as a rule, nearly the same. They are,

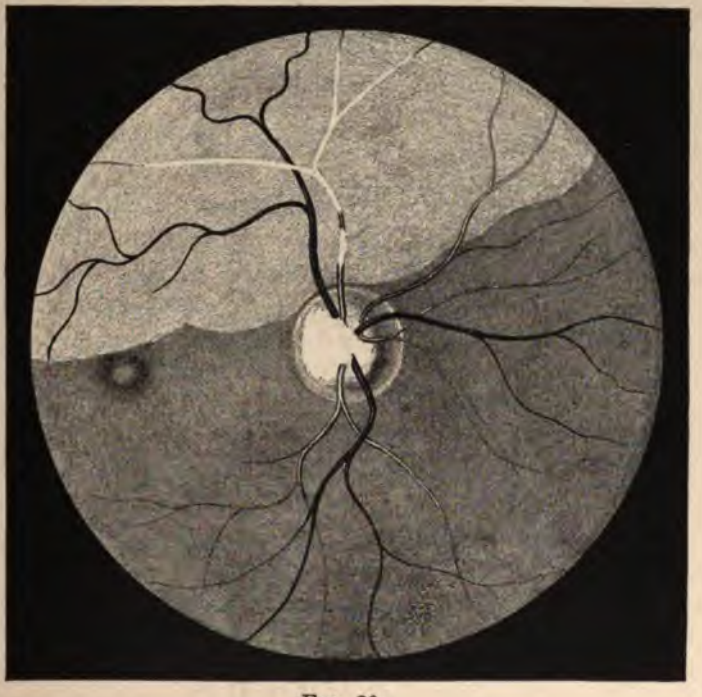

FIG. 20.

if anything, more conspicuous and more significant of the embolic process than where the main trunk is occluded, for the reason that, being situated more peripherically, they are more within the reach of the mirror. The place of stoppage is, however, usually upon the disk itself, and only in very rare cases in the retina, although such cases have been observed.

There is usually a small bulbous extension of the vessel, that is supposed to mark the seat of the embolic plug; beyond this the vessel

* "Trans. American Ophth. Soc.," 1882, p. 411.

becomes attenuated to a fine red thread for a greater or less extent. The sides of the vessel may be marked by fine white lines, or the entire vessel may give the appearance of being transformed into a thin white cord. The most remarkable example of this transformation is shown in the first case reported—that by Saemisch. This related to a gentleman who, upon waking in the morning, found that there was a defect in the vision of the right eye, much as if a screen had shut off the lower part of his visual field. He had used this eye only a few days before in shooting, and he was therefore sure that his sight was at that time perfect. Examination showed that the lower half of the field was wanting. The picture, Fig. 20, is taken with the inverted image, and the ophthalmoscope showed that almost the entire lower half of the retina appeared of a milk-white color, through which the choroid was dimly visible. The lower branch of the central artery presented, just below the disk, a slight cirsoid dilatation, below which appeared a sudden attenuation of the vessel, the whole of the artery below this point having the appearance of having been transformed into a white cord, with the exception that a short interruption occurred, where the red blood column could still be seen.

The central vision was normal, and was still so when the patient was last seen, six weeks later. The defect in the field remained, however, unchanged, as did the ophthalmoscopic appearances in the main, although the œdema into the retina had disappeared entirely.*

The optic disk in these cases usually becomes blanched in that portion of its surface which corresponds to the disturbance in the artery. The demarkation of this whitened portion is, however, not so regularly marked, or so restricted in its situation to the affected artery, as some writers appear to believe. This was so in the case just recorded, as the whitened portion extended over the greater part of the surface of the disk, both above and below, and the porus opticus and the place of emergence of the vessels were covered up by what seemed to be an exudative mass.

As a rule, the vision is suddenly impaired or even totally lost for a moment. This total blindness usually passes away more or less rapidly, leaving a quadrant-like defect in the upper or lower half of the field, according as it is the upper or lower branch of the artery that is affected.

Ordinarily, these cases of embolism of a branch of the central artery are accompanied by embolic processes in other parts of the body.

When, according to Dr. Knapp, a primary branch of the artery is

* "Zehender," 1866, p. 32.

affected, there is either a superior or inferior hemiopia, but when a secondary branch only is obstructed, a sector-like defect is observed in the upper or lower half of the field. The limitation, moreover, occurs in such a way that one border-line of both the hemiopic and sector-like defects coincides with the horizontal meridian.*

Dr. Knapp reports, among several of these cases seen by him, one in which there was a stoppage of the lower nasal branch, that was accompanied by a rather profuse hæmorrhage which occupied the section of the retina that was supplied by this division of the artery.† Dr. Knapp was inclined to look upon these hæmorrhages as the visible expression of an infarctus, such as occurs in other parts of the body, and which took place according to the theory then lately promulgated by Cohnheim. If this was really an infarctus, due to the causes alleged by Cohnheim, the wonder is that it does not occur more frequently.

There is nothing to show in the cases reported by Knapp, Landesberg, and Hoffman that the hæmorrhage from the veins is not due to a primary disease of the veins themselves and the result of thrombosis of the central vein or its branches, in accordance rather with the views of Litten than those of Cohnheim; and I am more inclined to this view, as I have seen two cases in which there were all the symptoms commonly attributed to embolism of a branch, including the sector-like limitation of the field where the arteries were not reduced in size, and where later, when the acute œdema which surrounded the lower portion of the nerve had cleared up, there were gradually developed over and around the lower branch of the principal vein the well-known signs of perivasculitis.

Moreover, it would appear from the recorded cases that retinal hæmorrhages occur as frequently when the central trunk is obstructed as when this takes place in a branch. Thus, according to the compilations of Schnabel and Sachs, out of thirty-one cases of embolism of the central trunk there were fifteen cases of hæmorrhage, and out of seven cases of some lateral branch there were three where extravasations occurred. The percentage, therefore, is about the same in each case.

It is claimed, also, that in embolism of a branch an anastomosis may occur, so that the vessel may become moderately if not entirely filled.‡

Capillary Embolism and Thrombosis.—The stoppage of the smaller vessels and capillaries of the central system has been but little

* "Archives of Ophthalmology," vol. iii, No. 1, p. 38.

† *Ibid.*, vol. i, p. 64.

‡ *Ibid.*, vol. xiv, No. 2, p. 290.

commented upon as an ophthalmoscopic sign and symptom, although it has been often suggested, as would naturally be the case, that it must play an important *rôle* in certain forms of inflammation of the deeper membranes of the eye, such as the choroid and retina. This would be particularly apt to be the case in some forms of retinitis, such as the retinitis of Bright's disease and septic retinitis where plugging of the smaller vessels from septic emboli, including those of micrococcal and bacterial origin, have been noticed, microscopically at least, by many observers from Virchow in 1854 to the present day.

These are, however, to be classed rather as forms of inflammatory disease than as a simple destruction to the circulation, and will, therefore, be further dwelt upon under another heading.

Thrombosis of the Central Vessels.—Many years ago I reported five cases to the American Ophthalmological Society of sudden loss of vision in which the symptoms were so exactly like, or so nearly like those of embolism as to lead to this diagnosis; made not only by myself, but also by my colleagues. Further consideration, however, induced me to change my opinion, and the cases were afterward published as examples of vasculitis in which the vessels had become obstructed by clots of blood or fibrin formed at the place of obstruction—or, in other words, that thrombosis had occurred in the retinal vessels or those derived from them.* It was not until five years afterward that this view of the matter was corroborated by Angelucci, who, in a case which presented all the symptoms of embolism, found on section of the eye that the cause of the trouble was due to thrombosis of the central vein.

The following case—one of the five alluded to above and upon which the diagnosis of thrombosis was based—is given because it is supposed to embody some of the great principles of inflammatory action upon the vessels as seen with the ophthalmoscope, and because many of the elements which enter into its composition are still under discussion:

A. O., aged sixty-two, presented herself in December, 1869, with the complaint that she was "blind in one eye." She had always been remarkably healthy, and had never had any previous ocular trouble. Three weeks before this, while standing perfectly still, sight in both eyes became suddenly obscured by a dense cloud, which, as she expressed it, had a "quivering motion." This was unaccompanied by any pain, and the sensations which the patient experienced at the time were exactly those which she had previously felt when about to faint, which had occasionally happened to her in former years. The

* "American Journal of the Medical Sciences," April, 1874, p. 313.

cloud passed away from the left eye almost immediately, vision becoming at once as clear as ever. The vision in the right remained so much diminished that the patient could only tell light from darkness.

During the three weeks between the occurrence of the trouble and her visit to us vision improved a little at times, but the improvement was only occasional, and then very transient.

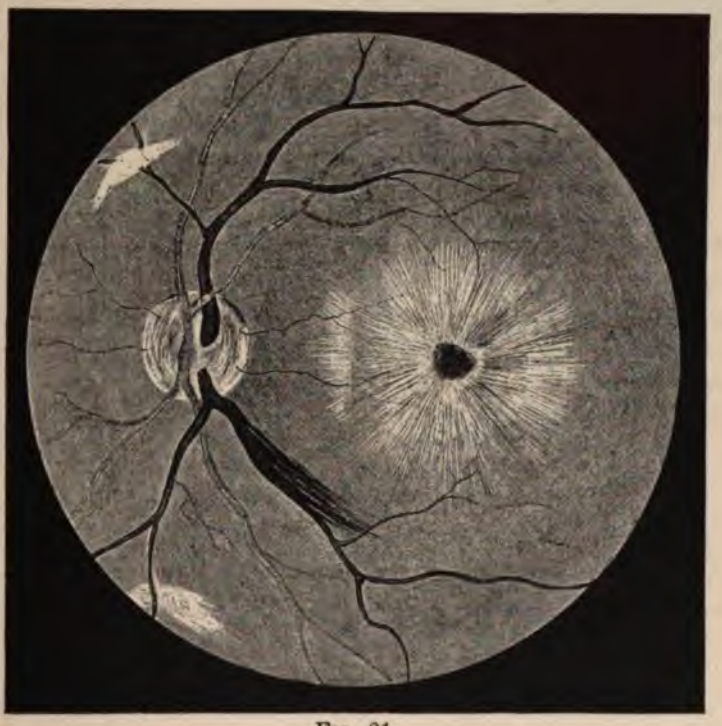

FIG. 21.

Externally the eyes presented nothing abnormal. The ophthalmoscopic examination showed that the media were perfectly clear. The optic disk was rather injected than pale, but not markedly so. The arteries were not much reduced in size, while the veins, on the contrary, were enormously distended, and were without any double contour from their very commencement up to their exit from the globe. There were three hæmorrhages, all of which were venous (see Fig. 21; also chromo-lithograph, Plate II, Fig. 1), one of which was evidently of recent origin, while the other two were manifestly of longer standing, as the hæmatin had been absorbed from them; they were, however, of no great date, as could be told by their color. The hæmorrhage of recent date was, as will be seen from the drawing, from one of the

principal branches of the central vein, and was close to the nerve; the earlier ones were both at the periphery of the field, one being above, the other below. There was no visible pulsation in either veins or arteries under as strong a pressure as it was thought safe to apply. The changes round the macula lutea were those which are commonly described in a case of embolism, modified, of course, by the lapse of time since the attack had occurred. The characteristic milky appearance extended still for a considerable circuit round the macula, and the bright cherry-spot at its centre was very pronounced, though it was larger than is usually the case. The left eye was normal in every respect, and vision with the proper glass ($+\frac{1}{8}$) was $\frac{20}{20}$; nor was there any limitation in the field of vision. An examination of the heart and kidneys showed nothing abnormal. The patient's bodily condition seemed to be better than the average at her time of life.

From the sudden and almost total loss of vision in one eye only, unaccompanied by pain or any marked general or local symptom other than a slight faintness, and principally from the ophthalmoscopic appearances, the cause of the trouble was pronounced to be of an embolic nature. The progress of the disease was as follows:

About a month after the first visit the large and fresh hæmorrhage had, together with the remains of the two earlier ones, been entirely absorbed, and the changes round the macula had disappeared. The veins were less gorged and had become somewhat spindle-shaped, being smaller at the nerve than at a short distance from it. The size and appearance of the arteries had undergone little or no change, and the vision had not improved at all. The patient was now dismissed, with the belief that further treatment would be of no avail, but was told to report occasionally for observation. In two weeks, however, the patient again returned, saying that she had been attacked with sudden and violent pain in the eye, and on examination it was found to be much injected, the pupil somewhat dilated, the bulb tense, and the media turbid. No view of the fundus could be obtained, and the supposition was that there had been fresh hæmorrhages into the vitreous followed by glaucomatous symptoms. Immediate operative interference was opposed. The following day the anterior chamber was partially filled with blood, but the pain and tension were less, and from day to day the eye appeared to be gradually recovering from the attack.

A month later, or two months after the first attack, the patient was again seized with violent pain in and around the eye, and when on the following day she visited us there were all the signs of an attack of acute glaucoma. As the eye was entirely sightless, as the

pain was intense, and as iridectomy could only be a slight palliative, it was determined to enucleate the eye. The operation was performed by Dr. Agnew, and the eye given to Dr. F. Delafield for examination, who kindly furnished me with the following description. As it is one of the very few eyes which has come to a microscopic examination in which embolus was indicated, I give Dr. Delafield's account entire:

"The eye was opened by an equatorial section, immediately after its extirpation. The cornea and lens are transparent. The vitreous appears transparent with the exception of two small red clots in it. The retinal vessels can not be seen. The optic nerve is white. At the macula is a dark-colored spot; in the fundus on the side opposite the macula is a long dark line, and near this a round dark spot. The eye was then hardened in potass. bichrom.

"After hardening, the vitreous still appears transparent, except at two points—a small spot over the pars ciliaris retinæ, and a larger spot at the equator. Both these opacities are caused by the presence of shriveled, colorless, red blood-globules, mixed with fine granules, situated in the vitreous close to the limitans hyloidea; otherwise the vitreous presents no changes. The iris appears normal; its vessels are for the most part empty. The ciliary body appears normal; the long veins running back to the choroid are filled with red blood-globules.

"In the retina the dark spots seen while the eye was fresh can no longer be distinguished. There are seen, however, at the fundus a small, irregular white spot and a white line. Both these appear to be due to the presence of a thin layer of fine granules (degenerated blood), adhering to the internal surface of the limitans hyloidea. The dark color of the macula is due to an increased pigmentation of the choroid beneath it. The rods and cones are badly preserved. The other layers appear normal, except that in the nerve-fibre layer there is an apparent atrophy of nerve fibres and increase of connective tissue, especially around the vessels. The vessels are all small, but filled with blood-globules.

"The choroid shows no change in the epithelial layer; the short and long posterior ciliary arteries are empty. Some of the large veins contain red blood-globules. Just behind the situation of the anterior clot in the vitreous, several of the large choroidal veins are filled with homogeneous, finely granular thrombi. The chorio-capillaris is empty. In the stroma of the choroid, between the vessels, are a number of little aggregations of lymphoid cells. These occur at many different points in the choroid. In the optic nerve the artery and vein are

nearly empty; the lamina cribrosa appears to contain an excessive amount of fibres and round cells."

The patient made a rapid recovery from the enucleation, and in a few weeks was dismissed from observation, able to use the remaining eye, in which vision was 1, to all intents and purposes as well as ever.

Nothing was heard of the case till about a year and a half after the operation, when I learned incidentally from the family physician that the patient had recently died. I heard that after passing from our care the patient had enjoyed good health, and had complained of nothing till within three or four months of her death, when she began to be troubled with peculiar sensations in the left leg, which at that time began to show signs of swelling with œdema, remaining, however, firm to the touch. The trouble in the leg gradually increased, extending from the foot up, until just before the patient's death it had assumed all the characteristic signs of phlebitis or "milk-leg." There were at this time physical signs of heart disease, which, as they were not present when the examination was made in regard to the trouble in the eye, were supposed by her physician to be secondary to the trouble in the leg, and due to some impediment in the circulation. Immediately after this the patient was seized with apoplexy and died.

The sudden loss of sight in the left eye, and the peculiar ophthalmoscopic signs in the neighborhood of the macula, point strongly toward what has been hitherto looked upon as embolism of the central artery. There are several things, however, which need to be taken into consideration. The milky whiteness at the macula and the cherry-red spot are not, as has been said, pathognomonic of embolism, for they occur in other affections.

A very strong argument against embolism is the condition of the vessels. It would, from the very nature of the case, be expected that, in occlusion of the central artery by an embolus, not only the main trunk, but all the branches would be either wholly empty or very much reduced in size; such, indeed, was the fact with Graefe's famous case, which led to the discovery of embolism, and so it has been with all the cases, with few exceptions, since reported.

Indeed, this has been always looked upon as the most prominent diagnostic mark of the affection. It was very different here, for, although the arteries did not appear as full as they should in a perfectly normal eye, there was not only no marked reduction in size, but there was even reason to believe that the apparent want of diameter was due rather to contrast with the abnormally increased veins than to any actual diminution in the size of the arteries themselves. It is very true that the arteries might have regained what was nearly

their normal size by the circulation being re-established, between the time of the attack and the first examination, which was made, it will be remembered, nearly three weeks after the seizure. This would be ample time for the restoration of the circulation. This might take place either through the natural passages or through an anastomosis, as has been alleged to occur by some observers in those cases in which the vessels do not show any material reduction of size. The condition of the veins differed too from what is usual in embolism of the central artery, for in the cases hitherto reported marked stasis has been absent. Here, on the contrary, great impediments to the venous circulation, with great engorgement of the veins and subsequent rupture, are very prominent symptoms.

The history of the case, both before and after the attack, seems to be against embolism, or at least different from what would lead us *a priori* to such a diagnosis. The patient had never been subject to rheumatism or any other disease which would lead to a favorable condition for the production of emboli. The heart was perfectly normal at the time of the attack, and remained so long after the patient passed from treatment.

The progress of the disease, though similar in its early stages to embolism, was very different from it later on. It will be remembered that, like embolism, the attack was ushered in by sudden blindness, and that the ophthalmoscopic appearances were in some respects similar to, if not identical with, those usually attributed to that disease.

Further, the œdema in the retina, as is usually the case, cleared up, the hæmorrhages were absorbed, and the veins regained almost their normal condition, the last examination showing in fact that, ophthalmoscopically, the fundus differed but little from a healthy eye. Here, however, the resemblance stops, for the eye, instead of remaining quiet, as has hitherto been the case with embolism, underwent a second attack, the conjunctiva and subconjunctival tissue became inflamed, there was intense pain with bleeding into the vitreous, hardness of the globe, and all the symptoms common to a glaucomatous attack, the hæmorrhages this time evidently being from the choroid and not from the retina. Was this second attack due to a second embolism, and the third to a third?

This is certainly not the clinical history of embolism of the central artery, and if it was not embolism, what was it? What other disease could have produced sudden total blindness with this train of symptoms? Certainly not simple retinitis hæmorrhagica. Nor does it appear at all likely that any form of neuritis or retinitis could so suddenly develop itself as to destroy vision in a single moment; such

is never the case. The complete and instantaneous blindness could hardly come but from one cause, and was no doubt due, from its very nature, to a sudden arrest of the circulation. This being the case, what other disease excepting embolism could produce the stoppage? And the answer is, phlebitis with thrombosis.

Might not this attack in the eye be the first expression of a coming, and more general, trouble in the minute branches of the circulatory system at large? A trouble which showed itself first by an impediment in the retinal circulation relieved by subsequent hæmorrhage, to be followed by precisely the same condition in the choroid, to be followed again by the same condition in the circulation of the lower extremities, to be followed in its turn by the same condition in the circulation of the head.

It may be asserted that all the phenomena presented in the different parts of the body may be explained by successive emboli occurring in the various regions affected. This may be true, but, so far as the eye is concerned, the condition of the vessels was not such as is usually described with embolism, for the artery, instead of being empty, was as full as in a state of health, and this could hardly have been brought about either by a collateral circulation or by regurgitation of venous blood. The condition in the lower extremities was one rather of a chronic or slowly progressing inflammatory action than the sudden stopping of the circulation by an embolus. Moreover, at the section of the eye no embolus was found in the central artery, and thrombi were found in the choroidal vessels.

Finally, the only symptom which seems to be absolutely pathognomonic of embolism, and of that only, is the *sudden blindness*. Is, then, sudden blindness always due to embolism? and might not this be explained as the final result of a series of processes which had been smoldering for some time, and which had ended in a stoppage, or at least in a reduction, of the circulation? This sudden shutting down of the circulation occurs in other parts of the body, and why not in the eye? A rapid transudation of serum from the capillaries and small vessels might so interfere with the fluidity of the blood as to render it unfit for circulation, or the stoppage might be brought about by some antagonistic condition of the walls of the vessels themselves. In a membrane so delicate as the retina, and one which the simplest experiment proves is so absolutely dependent on the circulation for the performance of its function, the slightest disturbance might serve to overthrow the equilibrium of the circulation, and thus bring on, just as Donders's experiment does, almost instantaneous blindness. Fox and Brailey have since reported a similar case in

which thrombosis is supposed to have resulted from primary inflammation in which there was microscopical evidence of thrombosis of two veins.*

The following case would seem to me to be one of this nature and to represent an example of acute œdema into the retina associated with an arrest of the circulation—a transudation which was probably favored, if not caused, by a diseased condition of the vessels:

J. B. D., aged forty-seven, physician, at two years of age had a severe attack of acute rheumatism, by which all the joints of the hand were enlarged and several anchylosed. At the age of nineteen he had another attack of rheumatism, which laid him up for three years and left him a "perfect wreck." The patient has never had, till very recently, any other disorder. In January, 1847, he first noticed a trouble in the eye. He was engaged in conversation, when a black cloud passed over his left eye; closing the right, he found he was perfectly blind in the left. The blindness passed off in a minute or two, when vision became as clear as ever. Just prior to this attack he had severe pain over the left brow. After a week had passed the patient had precisely a similar attack without any pain, the eye becoming totally blind, and after a minute or two sight returning as good as ever. From this time forward until the present moment the patient has had repeated attacks of the same nature, sometimes daily and then again not for weeks or months. He became so accustomed to their recurrence that he paid very little attention to them, though when sitting quietly he would often cover the right eye and watch the sight return in the left. These attacks, since 1847, have been too numerous, he says, to "keep count of." Until within this year (1871) the patient's general health has been better than usual, and he has had no rheumatism. In February of the same year he had a severe attack of facial erysipelas, from which he suffered great pain. He recovered from this in due time, and was ready to leave his sick-room, when he was taken by a very sudden attack of dropsy. He went to bed apparently as well as usual, and on the next morning was astonished and alarmed at finding his belly immensely distended, and measuring fifty-seven inches in circumference. His urine was examined at the time; albumen was found, but no casts. Recovering from this, he gradually resumed his practice, and was engaged in this at the time of the present trouble, for which he consulted me in September, 1871.

Three days before this the patient was sitting quietly, talking, when the left eye suddenly became obscured. Thinking that the

* "Ophth. Hosp. Rep.," vol. x., Part II., p. 205.

sight would return again, as it always had, the patient paid very little attention to it. But on this occasion the sight did not return till the following day, and then only in a reduced degree, and only for a moment, when it suddenly left him again. Becoming alarmed, he consulted an oculist, who, from the suddenness of the blindness, and from the peculiar ophthalmoscopic picture, pronounced it a case of embolism, although there was no reduction of the calibre of the vessels, which might be explained on the ground of an anastomosis having taken place.

When seen by me, vision was reduced to quantitative perception of light in a small portion of the upward field. The ophthalmoscopic

FIG. 22.

picture (Fig. 22, also chromo-lithograph, Plate II, Fig. 2) was the most remarkable that I have ever seen. At first sight the entire retina seemed to have a milky-white appearance. The outlines of the optic nerve were obliterated, and its position only made apparent by the convergence of the vessels at the porus opticus. On closer inspection, however, a faint red reflex of the choroid could be got at the lower periphery of the fundus, and at the macula was the characteristic

bright cherry-colored spot. The vessels were of the usual size, and there was no sign of any interruption in them, or reduction, in any place, in their diameter. On the contrary, while the arteries appeared of their usual dimensions, the veins, if anything, seemed to be distended.

There was no light streak visible on any of the principal trunks or any of the branches. The œdema was so great as to give the appearance of red vessels running on a white ground.

An examination of the urine showed a slight amount of albumen but no casts, and there was, according to an expert who examined the heart, "an obstruction at the aortic orifice and consequent hypertrophy of the left ventricle, without, however, regurgitation at the aortic or mitral valves, and without dilatation."

With such symptoms, and with such an ophthalmoscopic picture, the diagnosis of embolism would, of course, be the first to suggest itself. But here, as in the other case, there are weighty objections against it.

In the first place, if it were embolism, how could it be possible that precisely similar attacks, so far as the rational signs were concerned, had occurred over and over again for a long series of years, and were at one time of weekly or even of daily occurrence? Attacks which, so far as the patient could tell, only varied from the last one in the fact that the blindness invariably disappeared after a short interval instead of remaining, as in the present instance, permanent. This could only happen in these "almost countless" attacks under the supposition that the plug had rested for a moment, stopping the circulation, and had then been dislodged so as to allow the circulation to be entirely restored. Almost absolute blindness could only be brought about, if due to embolism, by a plug in the trunk of the central artery, and the only place it could be dislodged from, so as to permit the circulation to be restored, would be from the very mouth of the central artery, just where it is given off from the ophthalmic. Once fairly within the orifice, it could only be driven farther and farther into the eye, as there is no collateral and anastomosing branch sufficient to receive it, and the blindness would, as is usual in cases of true embolism, be permanent. It would certainly seem to pass belief that such a thing could occur over and over again, for the space of twenty-five years, and be due to a real embolism. Thus the explanation hitherto given for the restoration of vision by the dislodgment of the plug is not applicable here, if it ever were.

Then, again, the condition of the vessels only twenty-four hours after the attack is against embolism, as no reduction in size was appar-

ent, and the explanation of this fact by supposing that the arteries had regained their normal diameter through an anastomosis, in so short a time, seems to be untenable either from their actual appearance or from their subsequent behavior. For we would suppose that, if the current had been restored through anastomosis, the longer the interval after the attack, the fuller the vessels would become, and that if, as in this case, the anastomosis had been sufficient to establish the normal size of the vessels, this would have been maintained. Such, however, was not the case, for the vessels began to gradually

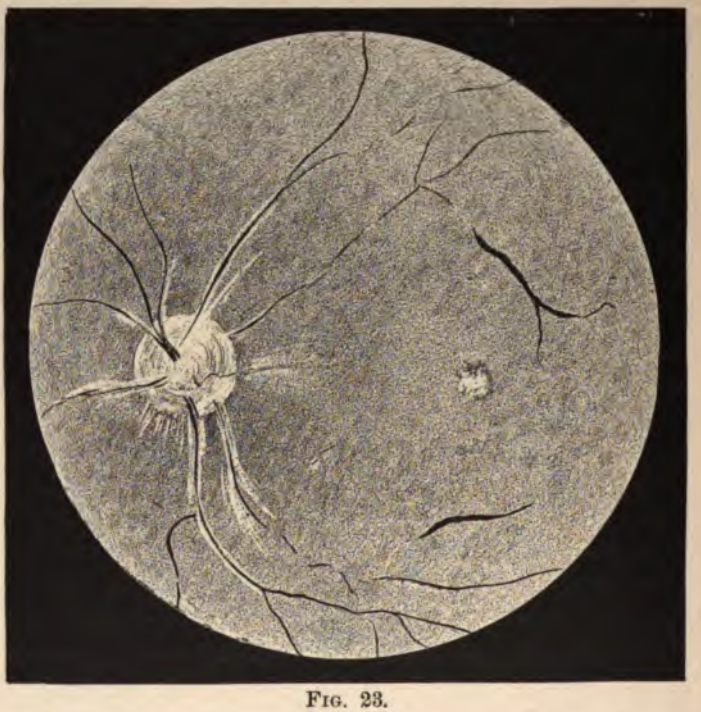

FIG. 23.

diminish from week to week till in about six weeks after the attack the secondary branches had, in some places, faded away, or had become of thread-like thinness, while along the principal branches longitudinal white bands were formed, and there was a hyperplasia of connective tissue in and around the nerve, which, together with the retina. passed gradually into a state of atrophy. In two places a thread-like vessel could be followed from one of the larger branches of the upper and lower veins for a considerable space, when it very rapidly increased in size for a short distance to become again suddenly filiform, and then untraceable. (Fig. 23, also chromo-lithograph, Plate III, Fig. 1.)

As the rest of the vessels were either empty or nearly so, these distended portions were supposed to be blood coagula within the walls of the vessels.

It is very evident, from the white bands running along the vessels, and their subsequent atrophy, that the vascular system itself was severely affected, and that the signs above alluded to were the results of vasculitis and perivasculitis, in which the veins were principally affected. Now, whether this was secondary to a stoppage in the circulation by an embolism in the artery, or whether the condition of the vessels themselves was the cause of the arrest, is the question. I am inclined to the latter view, as already pointed out, from the behavior of the vessels.

It would not appear that either the retina or choroid was primarily affected, but that the changes which took place in them were secondary, since there were none of the active signs of inflammation in either the retina itself or in the nerve; there was no swelling or congestion of this latter, nor plaques or swelling in the retina itself. Besides, instantaneous blindness would not be apt to come from a simple retinitis or neuritis.

The most striking feature of the whole case was, however, the pronounced œdema, which, intense as it was, had the appearance of being, and no doubt was, due to a very profuse and rapid transudation of the watery contents of the vessels into the surrounding tissue; and the idea at once suggested itself, from the fact that the patient had had so sudden and extensive an effusion into the peritoneal cavity, that perhaps a similar condition of the vessels had produced a similar result in the eye; and that, secondary to the obstruction at the heart, there might have sprung up some morbid condition of the walls of the minute vessels and capillaries antagonistic to a free and rapid circulation of the blood, and favorable to a profuse exudation of serum into the surrounding tissue, should an arrest of the circulation take place from any cause. This sudden arrest of the circulation takes place in other parts of the body, and why not in the eye?

Might it not happen that, just as, in the preceding case, there had been an active phlebitis in the eye followed by that in the extremities, so in this case there had been a phlebitis of a lower type with no hæmorrhages, but with passive exudation followed by secondary atrophy, with the signs of intra and perivasculitis? Or it might be that the œdema was the primary cause of the trouble, and that from a morbidly relaxed condition of the walls such an extensive exudation had taken place that recovery was impossible before secondary changes took place. May not, after all, the heart be the seat of the true cause

of not only this last, but of the preceding temporary attacks of blindness? It will be remembered that there was obstruction at the aortic orifice, and in a feeble and marasmic patient might it not occur that for a moment or two the action of the heart should be so reduced that it could not force the blood through the obstructed aortic orifice with sufficient power to reach so distant an out-post of the general circulation as that of the retina, in which any reduction of the blood supply is at once followed by a corresponding degree of blindness—so much so that a little external pressure on the globe will produce it? So too in syncope, one of the earliest symptoms is a rapid obscuration of sight. Anæmia of the retina will invariably produce sudden blindness, and there may have been some anatomical condition which would make it, in this case, more difficult for the heart to force the blood into the left eye than the right, and hence would account for the recurring blindness always taking place in the left and never in the right.

Supposing a momentary reduction of the heart's beat did take place, and then, after a few feeble efforts, the heart recovering its tone, the circulation was restored again to its natural force, vision would then return, just as it did in this case, over and over again. But how can this fact be explained by embolism?

And, finally, may not this last attack, in which the blindness became permanent, be similar in cause as it was in effect to the preceding ones, and be but an exaggerated expression of precisely the same condition?

It might well be that for a moment little or no blood from a more pronounced reduction of the heart's beat passed into the central artery of the retina, and that, this being a terminal artery, there was a reflux from the veins into the arteries as soon as the pressure was taken off from behind; and that the circulation, instead of being restored at once as in the other cases, was not re-established till secondary processes had been set in progress, which not only maintained the temporary and functional loss of vision, but which led to organic changes with permanent loss of sight.

Since the publication of this case Nettleship has also called attention to two similar cases, characterized by repeated paroxysmal failure of sight in connection with heart disease. In the first case there were transient attacks of loss of vision in the right eye, followed by permanent blindness, with the ophthalmoscopic signs of arterial blocking. Subsequently there were many temporary attacks of loss of vision in the left eye, with contraction of the visual field. There was mitral stenosis.

In the second case there was blindness of the left eye, with changes, the result of retinal hæmorrhage and thrombosis. Afterward there were repeated attacks of blindness of the right eye and contraction of the visual field, with small opacities in the vitreous body, spontaneous arterial pulsation, and severe aortic disease.*

The first case reported by Angelucci, in which a thrombus was found on section in the central vein, was as follows:

A young man of twenty-three became suddenly blind in the left eye while at work. He was suffering from stenosis of the mitral and aortic valves. His other organs were sound.

The ophthalmoscopic examination of the left eye, in which the blindness was, showed that the borders of the disk were somewhat irregular and of a moderately white color. The arteries were narrower than normal. The veins were dilated and somewhat tortuous for their entire extent. There were no retinal hæmorrhages in this eye, in which there was no perception of light. In the right eye the vessels were, upon the whole, of normal calibre. In immediate connection with a small arterial branch which ran toward the temporal side there was a small triangular hæmorrhage, which was the only one present in the entire fundus.

The autopsy showed a spot of softening in the cortical substance of the left hemisphere, dilatation and hypertrophy of the left side of the heart, with signs of endocarditis, stasis in the vessels of the liver and infarctions in the spleen, and hyperæmia in the alimentary canal.

Microscopical Examination.—That of the left eye (Fig. 24) showed that the central vein was somewhat dilated and closely embraced the artery. At the outer border of the lamina cribrosa, and about one millimetre from the retina, the vessel was entirely closed by a thrombus which, in its unstained condition, consisted of a strongly glittering fibrinous plug (Fig. 24, *Th*). This was attached so firmly to the intima of the vein that a sharp limitation between the two could not be detected.

The artery was seen to be full of blood. There was a copious exudation in the neighborhood of the thrombus, which was made up of a dense collection of lymphoid cells and red blood-corpuscles (Fig. 24, *I*). This accompanied the central vessels only for a short distance (0·5 millimeter), but stretched out here and there in different degrees of intensity between the fibres of the optic nerve. The thrombus had the appearance as if it was beginning to become organized. Fig. 25 shows a cross-section. There was nothing abnormal in the walls of the artery, with the exception that the muscular coat was somewhat

* "British Medical Journal," June 14, 1879, p. 889.

thickened. This cross-section also contained blood. The wall of the vein, on the contrary, was three times as thick as normal, and formed

FIG. 24.

a broad ring 0·06 millimeters in diameter (Fig. 25, *V*). Whether this thickening was due to a pre-existing chronic inflammation or to acute œdematous swelling could not be told. It was, however, apparently due to the former.

There were, moreover, signs of acute inflammation, not only in one but in several places, especially where the veins emerged from the nerve. Here were found congregations of round cells, both in the wall itself and in the perivascular lymph spaces which were here and there dilated, and also beyond them in the surrounding tissue of the vessels. This inflammatory infiltration extended into the smaller venous twigs and those of a capillary character.

The endothelium of the veins was intact throughout (Fig. 25, *p v*). Some of the smaller vessels of the retina were enlarged, but, besides this, there were no other changes, and there were absolutely no hæmorrhages or remains of pigment. As a consequence of a disturbance of the circulation, the first thing to be noticed was the atrophy of the papilla and optic nerve. The single nerve bundles, as can be plainly seen in the transverse section, were surrounded by a broad lymph space (Fig. 25, *L*), and the lymphatic sheath of the entire nerve was considerably thickened.

It follows directly from the examination that the veins were affected by a primary disease, and that the changes were due to phlebitis and were not simply a consequence of the thrombus. This fol-

lows, also, directly from the examination of the fellow-eye. In that eye the exophthalmoscopic examination revealed some hæmorrhages which were shown afterward to be situated in the external granular layer. There was, however, no thrombus found at all in this eye, so that the inflammatory symptoms could not have been excited by a thrombus. There was, however, a pronounced phlebitis, with thickening of the walls of the vein and aggregations of round cells. This phlebitis, however, did not extend to the entire circumference of the vessel, but limited itself to single small sections of it. The process in this eye was precisely like that which has been described as taking place in the left eye, only less in degree, probably because it was examined in an earlier stage of the disease.*

Here we have a most remarkable condition of circumstances, namely, a patient with heart-disease, endocarditis and valvular softening—the very subject for embolic processes. With this we have the sudden blindness accompanied with the ophthalmoscopic signs usually attributed to embolism, and yet the autopsy shows that not only was there no embolus present, but that the arteries were not even diseased, while in the second case examined by Angelucci a thrombus

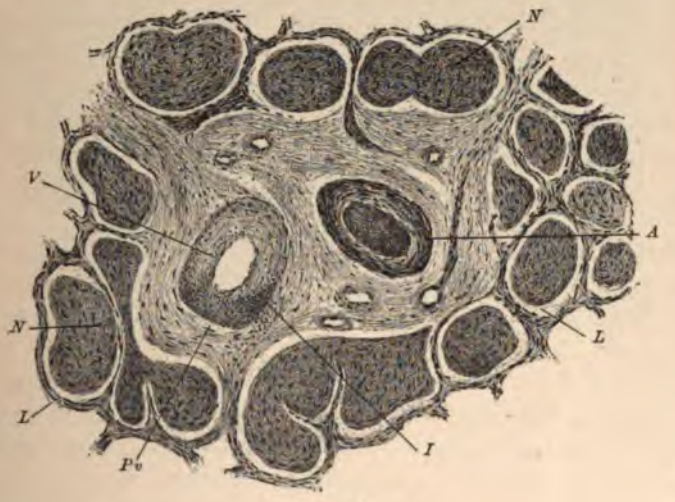

FIG. 25.

was found in the central vein before its entrance into the lamina cribrosa, and the veins were diseased.

All this is most important, and I would refer my readers to a careful perusal of the original.

* Zehender, 1878, p. 443.

This case would, moreover, seem to me to be a perfect corroboration of the correctness of the diagnosis of thrombosis applied by me to the cases published some few years prior to the microscopical investigations of Angelucci.

In 1878 Michel published some cases of thrombosis of the central vein which differed from those reported by myself and Angelucci in the fact that the phlebitis was of a more pronounced degree and the ophthalmoscopic symptoms were of a more inflammatory character.

Out of the seven cases reported by Michel the trouble occurred in only one eye with the exception of a single case. The patients were all between fifty-one and eighty-one years of age, and in all, without an exception, there was sclerosis of the peripherical arteries. In many there was emphysema, and in a few hypertrophy of the left or right ventricle.

The attack was always of a sudden nature, the blindness usually coming on over night, with no prodromes, and in the beginning with no subjective symptoms.

In general, the commencement of the disease shows a great similarity to embolism, but the vision is never so completely lost, and has a greater tendency to improve in the course of the disease, although only in a temporary manner. The loss of vision is in proportion to the development of the anatomical cause, and the improvement in vision is also in proportion to its removal, and when this takes place sight often improves to a degree that is out of proportion to the ophthalmoscopic appearances which for a long time may remain the same.

Michel recognizes three degrees of the trouble: 1st, A complete closure of the central vein. 2d, An incomplete one. 3d, One of so trifling a degree that simple stasis in the venous system only results. One of these degrees can pass into another, and the two first can be formed from the last.

In the first intensity the optic nerve and surrounding retina appear filled with suffused blood. The contours of the nerve are indistinct, and there are sometimes, besides the sanguinary suffusion, isolated hæmorrhages of a tongue-like character which follow the striated direction of the nerve fibres, and are situated at a little distance from the disk (Fig. 26). Near the nerve both arteries and veins are lost to view; beyond this the venous branches so predominate in their conspicuousness over the arteries that these latter are hardly to be traced.

The principal veins are very tortuous, and in certain sections have a sausage-like appearance, and they are about double their usual size

and have a blackish-red color. This dark color of the blood-column is sometimes interrupted in its course, so that one gets the impression of a longer blood-clot, which is broken in one or more places by fluid blood. The diameter of the vessel is increased at this spot, so that broader sections alternate with narrower ones. Occasionally the interruption is complete, and this portion then shows a yellowish-white color, which is to be looked upon as a collection of white blood-corpuscles. There are also often signs of a grayish discoloration of the retinal tissue in the neighborhood of the vessels, and these latter are at times accompanied by white stripes.

FIG. 26.

In the region of the macula there is usually at a very early date a grayish or yellowish white discoloration with a red spot in the centre, which is to be looked upon as an actual hæmorrhage, since it very soon undergoes changes in form.

The second degree, where the stoppage is incomplete, is like the first, only less intensified. The hæmorrhages are more striated and are also situated within the surface of the disk, while the vessels can be traced to the porus opticus.

In the third degree, or that in which there is only simple stasis, the extravasations are few, and the most conspicuous feature is the

great discrepancy between the size of the veins and that of the arteries. The veins are much dilated, tortuous and of a deep and dark-red color—much more so than is ever noticed in venous stasis, in consequence of inflammatory changes in the nerve. Besides the few ordinary hæmorrhages of a tongue-like character, there are small round hæmorrhages in the periphery of the field. There are very few or no changes at the macula.

In these cases either the thrombus may become organized and the lumen completely closed, or the lumen may be either partially or entirely restored.

When the closure of the vein is complete, the diffuse red color of the nerve and surrounding region undergoes a partial resorption. The edges of the nerve become a little clearer. The ecchymoses get smaller. The veins can be traced nearer to the disk, but always maintain their dark color. The arteries, like the veins, become more visible and more tapering as they approach the disk. Secondary hæmorrhages may now occur with concurrent loss in vision. The vitreous becomes clouded by a diffuse opacity, and what are known as retinal strings make their appearance in the tissue of the retina.

In the second intensity of the trouble new extravasations take place just after a better filling of the vessels could be proved to have occurred with the ophthalmoscope. The ophthalmoscopic appearances become less marked, the hæmorrhages are somewhat absorbed, and become of a light-yellow color. This discoloration shows itself first and strongest in the centre of the round ecchymosis, so that there is a yellowish centre surrounded by a hæmorrhagic periphery of a blood-red color. This yellowish centre is probably due to fatty degeneration. This change also takes place in the smaller extravasations to such a degree sometimes that all the region between the nerve and macula is filled with small yellowish spots. At the macula there often appears a larger hæmorrhage of an irregular shape, and the entire region of the yellow spot is marked by a grayish infiltration. There is no swelling of the nerve.

There is certainly a strong resemblance between the picture here described and that which is found in some forms of neuritis, especially neuritis descendens. There is this difference, however, that the arteries are very narrow, sometimes threadlike, while the veins are excessively dilated and contain a very dark blood. The veins seem, moreover, to be cut off abruptly in their course, as if they suddenly entered into untransparent tissue (Fig. 27), and then after a while just as suddenly appear again. At all these places there is an actual upheaving of the tissue of the retina, so as to produce a rolling surface

with alternate elevations and depressions. A small number of punctiform and flocculent opacities are found in the vitreous.

In the third variety, where simple stasis occurs and where the lumen remains open, perfect resolution may take place, and the ex-

FIG. 27.

travasations may be absorbed without leaving any trace. The contours of the nerve become sharply defined again, and there only remains a slight discrepancy in the size of the vessels, the arteries being a trifle smaller and the veins a trifle larger than normal and of a deeper and darker color. But it must be remembered that the disturbance to the circulation, although a temporary one, always leaves the disk whiter, which after a time appears of an atrophic character. The vision always remains somewhat, oftentimes considerably, impaired. The intra-ocular tension is never increased in any of these varieties.

Microscopical Examination. — The eye examined by Michel showed that there was a thrombus, which was completely organized, in the central vein. This was situated at about six millimetres from the entrance of the optic nerve into the bulb, and extended up to within three or four millimetres of the exit of the vein. The arteries

showed only a slight increase in the adventitia in breadth, the intima was intact, and the lumen was entirely free (Fig. 28 A). There was, on the other hand, complete closure of the vein at the spot where the thrombus was formed (Fig. 28 T.) The lumen of the vein was filled out with a mass which was rich in nuclei, protoplasmic aggregations containing large nuclei, and white blood-corpuscles. The mass, where it touched the vein, was for the greater part adherent to its inner wall (Fig. 28 V).

The walls of the vein were moderately thickened, and the thrombus extended for about one and a half millimetre.

The connective tissue, upon the inside of the central canal, instead of being open and loose, was very thick and close. The nerve-fibres were atrophic. The vitreous was much changed, and there were many vessels of new formation, and membranous strings.

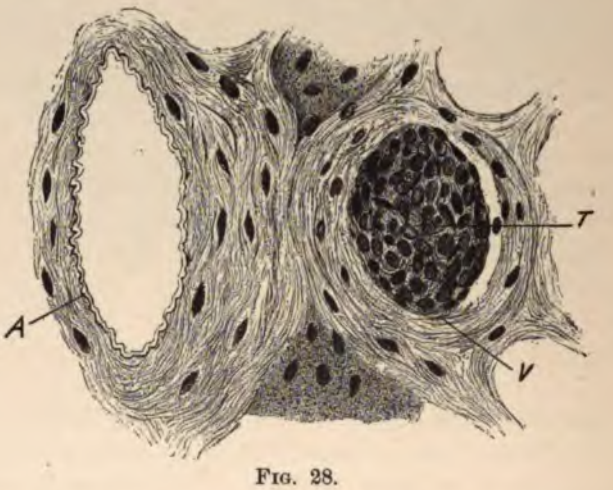

FIG. 28.

The thrombus was probably due to the hypertrophy of the endothelium of the vein, but whether the change in the inner wall of the vessel was the first step, or whether there was a primary coagulation which caused an irritation to the wall, and through this a proliferation of the endothelium, could not be definitely determined.*

Thrombosis of the Central Artery.—So far as I know, there has been no case of pure thrombosis of the retinal artery which has as yet been admitted to be such under microscopical investigation. All the cases of occlusion of the artery have been declared hitherto—by their authors, at least—to be due to embolism, in contradistinction to throm-

* "Archiv. für Ophth.," Band xxiv., ab. ii., p. 37.

bosis, but it must be admitted that, *a priori*, there is no reason why thrombosis should not occur, and very probably some, if not most, of the cases which, on section, have been reported as embolism have been due to thrombosis—notably those by Sichel, Nettleship, Schnabel, and Sachs.

In other words, I am still of the opinion that I held some dozen years ago, "that, in the train of symptoms classed under the title of embolism, too much significance has been laid upon the stoppage of the circulation by the importation of a plug formed at a distance, while not enough stress has been laid upon the mechanical actions regulating the supply of the blood within the eye, as well as upon the conditions of the walls of the vessels themselves, and their contents." *

Priestly Smith, partaking of this opinion, has lately laid down the following data for a differential diagnosis between embolism and thrombosis:

In thrombosis we have:

(*a.*) Previous attack of transient blindness in the blind eye.

(*b.*) A simultaneous attack of transient blindness in the fellow-eye.

(*c.*) Previous or subsequent attacks of transient blindness in the fellow-eye, especially if the conditions of the onset were the same in the permanent as in the transient attacks.

(*d.*) Signs of disturbance of the cerebral circulation at the onset of the blindness, giddiness, faintness, and headache.

In embolism we have:

(*a.*) Absence of transient attacks of blindness, either in the blind or in the fellow-eye.

(*b.*) Absence of giddiness, faintness, or pain at the moment of onset.

It must be admitted that the distinctions here made are not very numerous or very decisive, but at any rate they will serve to call attention to a supposed difference in origin until more essential and more characteristic differentiations can be made by future anatomical investigations.

Thrombosis from Uterine Disorders.—That the puerperal state and menstrual irregularities occasionally gave rise to either transient or permanent loss of vision was known even in pre-ophthalmoscopic times. It has usually been attributed, even in later times, to some central origin, or to retinitis albuminurica, or retro-bulbar neuritis; but that it may be caused by arrested retinal circulation and thrombosis within the eye, the following case would go to show. It forms

* "American Journal of the Medical Sciences," 1874, p. 328.

one of the series already alluded to as published, in which the physical and rational signs resembled those of embolism.

A. B., widow, twenty-six years of age, has always had ordinarily good health, but has never been very robust. Has always menstruated regularly. The last time that this occurred the flow continued only two days, instead of for one week, as was usual with her; at the same time she was suffering somewhat from constipation. On the day following the appearance of the menses she was sitting, in the evening, looking at the gas-light for some minutes, when suddenly a dark cloud passed over her right eye, and, on closing the left, she found herself entirely blind. After a little while, however, a strong perception of light returned, which was followed, after a time, by the ability to see very large objects, very indistinctly, however, and only when placed far to the right. On examination, both eyes appeared externally per-

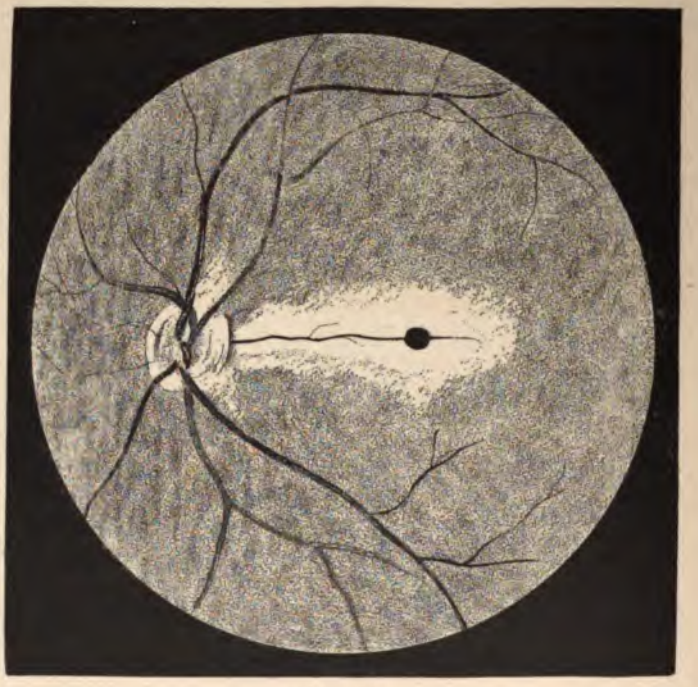

FIG. 29.

fectly normal, as was, indeed, the left, in every respect, with vision = 1. The right eye had merely perception of light. The ophthalmoscopic examination of this eye showed the media to be perfectly clear, the optic disk beyond being, perhaps, a trifle more congested

than usual, appeared normal, as did the retinal vessels, neither veins nor arteries showing any marked deviation either in size or course, except that the light-streak was reduced in brilliancy, and near the

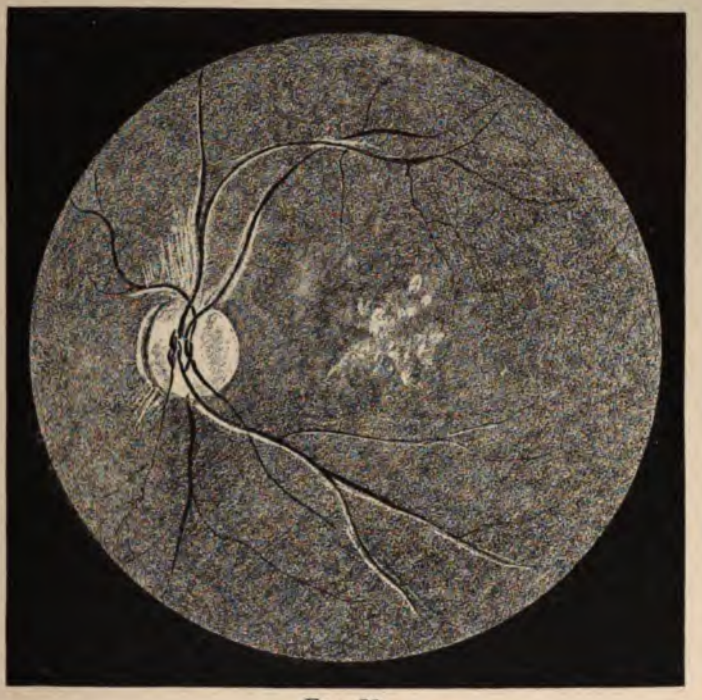

FIG. 30.

disk entirely absent. From the outer edge of the disk, however, straight out to, and including, the macula, ran a grayish-white patch, which had the appearance of a dense exudation into the retina.

The position of the yellow spot was marked by a perfectly circular bright-red spot.

Three or four days after this the patient again presented herself, and a careful examination into the heart and kidneys showed that these were normal. The ophthalmoscopic appearances had undergone a slight change, for, while the form of the exudation remained about the same, its color had changed from a grayish-white to a yellowish-white, while from the edge of the disk, and straight through the centre of the effusion and through the red spot at the macula, ran a small vessel, which had been developed since the examination four days before, and was most probably a simple increase in size of a pre-existing vessel. (I have since thought that this enlarged vessel might have been a cilio-retinal vessel, or one of the vessels which

come down along the sheaths of the nerve, and that it might be looked upon as an attempt to establish a collateral circulation.) It was at this period that the picture was taken. (See Fig. 29; also chromo-lithograph, Plate III, Fig. 2.)

The exudation was gradually absorbed, while in the region of the macula spots which appeared to be due to fatty degeneration began to make their appearance, and the vessels, from having their normal calibre, gradually fell away in size, while longitudinal white bands began to develop themselves along their larger branches, until on March 28, 1871, or about a month after the attack, a general atrophic condition had taken place, with the ophthalmoscopic appearances shown in Fig. 30; also chromo-lithograph, Plate IV, Fig. 1.

The points of interest in this case are, first, that the attack came on during a scanty menstrual flow, suggesting the inquiry whether the trouble in the eye had not had some connection with the disturbed condition of the uterus; just as we often see retinitis hæmorrhagica from suppression of the menses, especially in women about the period of change in life. The fact that in this case no hæmorrhage took place does not necessarily imply that stasis did not, while, on the other hand, it might be the very reason why the attack was so serious and followed by such disastrous results, there being no relief to the circulation till processes had been set in motion which were not arrested after the circulation had been re-established. The second, and most interesting point, is that the vessels were not reduced at the first examination, a few days after the attack, but that they *gradually* became so during the time she was under observation, which certainly would not have been the case had the trouble been due to embolism, and the normal distention of the vessels due to an anastomosis.

I have since seen several similar cases, one of the most interesting of which was one where I was inclined to think no actual thrombosis had been formed, but where there had been simply an arrest of the circulation for a time, which was afterward removed. The patient was a middle-aged woman who was at the time nursing an infant of about one year old. The day before she had quite suddenly lost the sight of the left eye, the vision of which was, at the time of the examination, so defective that she could only tell light from darkness. The right eye was normal in every respect. The catamenia were still absent.

The ophthalmoscopic examination showed the arteries to be considerably reduced in size, while the veins were enormously dilated and filled with dark-purplish blood. There was a minute hæmor-

rhage at the outer edge of the nerve, but no other extravasation anywhere to be seen.

Thinking that the attack was dependent upon the attempt of Nature to restore the menstrual flow, the patient was ordered to wean her child at once. Under leeches, frequently applied, with the bleeding kept up for a considerable time, and other derivative measures, the vision improved steadily for about three weeks, when it amounted to $\frac{2}{4}$. The venous congestion gradually disappeared and I imagined that the patient was cured, when suddenly, and just about four weeks from the previous attack, vision again left her. The same treatment was renewed, and vision began again to improve until it reached $\frac{2}{3}$, when it again failed, though to a less degree, in about four weeks from the previous date. Almost immediately after this the menses returned, and with this vision also, which then remained at $\frac{2}{3}$, and no further deterioration took place.

Priestly Smith has more recently reported some cases of the same nature, among which was one of much interest, as it seemed to be dependent on reflex action from the condition of the uterus. In this case temporary attacks of loss of sight were followed by a permanent blindness in one eye, caused, in Mr. Smith's opinion, by the arrest of the circulation and thrombosis of the artery.*

Hæmorrhage into the Sheath of the Nerve.—Dr. H. B. Sands first called attention to the above process as a possible explanation of the occurrence of sudden monocular blindness. The case was that of a young physician who suddenly lost the vision of one eye in which the ophthalmoscopic signs were those which are usually attributed to embolism. Vision was at first totally abolished, but later improved somewhat peripherically, leaving, however, a dense and permanent scotoma.

The theory which appeared to Dr. Sands to explain most satisfactorily the morbid phenomena in question was that which assumed the primary lesion to have been an extravasation of blood into the sheath of the optic nerve between the globe and optic commissure.†

Such a lesion had not at that time been demonstrated by dissection, but it has been since by several observers. Thus Priestly Smith has published a case, with a beautiful drawing of cerebral hæmorrhage, with passage of blood into both optic nerves. This was the result of a fall in which the patient, a mechanic, had slipped on the ice with but little apparent injury at the time, but which was followed some ten days later by headache and vomiting. At this time an oph-

* "Ophthalmic Review," vol. iii., p. 1, 1884.

† "Trans. American Ophth. Soc.," 1866, p. 2.

thalmoscopic examination showed that there was no optic neuritis. The patient died the following day. At the autopsy, on removal of the dura mater, a large quantity of blood escaped, which was found to have come from a cavity of the left frontal lobe, about as large as a walnut. The clot was confined to the left side of the skull, but extended backward to the cerebellum, both on the convexity and at the base.

Fig. 31.

The optic nerves showed bulbar enlargements behind the globes, and appeared from their color to contain blood.

The optic nerve, which had been previously hardened, was then frozen and divided by a longitudinal section. Within the distended nerve-sheath near to the eyeball there was a blood-clot, and the precise relations of this are of some interest, as they show in a beautiful manner the presence of the three sheaths, as described by Schwalbe—namely, the dual, arachnoidal, and pial (Fig. 31). The blood-clot lay entirely external to the arachnoidal sheath, while the subarachnoidal space had been injected with colorless fluid, doubtless the cerebro-spinal fluid forced into it from the subarachnoidal space of the brain by the increased pressure within the skull.*

It will be observed, however, on looking at the figure, that the pial sheath was still in close apposition with the nerve stem; and that neither the blood in the subdural space nor the fluid in the subarachnoidal space passed into or beyond the lamina cribrosa, which is in accordance with the effect produced by artificial injections. (See vol. i., p. 27.)

In the case above related there were no ophthalmoscopic changes,

* "Trans. Ophth. Soc. of the United Kingdom," vol. iv., p. 271, 1884.

and there is no mention of a microscopic examination as to the condition of the nervous tissue. In a case, however, reported by Dr. Silcock, in which no ophthalmoscopic examination could be made on account of the maniacal condition of the patient, microscopical examination showed after death that the nerve was inflamed and the disk swollen, so that it is still uncertain as to how far a distention of the nerve sheath, with blood or fluid, can be the cause of ophthalmoscopic appearances.* That it should provoke at times changes in the nerve which should be seen with the mirror is most probable. That it does not invariably do so is shown by the fact that flagrant cases of choked disk do occur where there is no distention of the sheath, while on the other hand distention of the sheath occurs where there is no choking of the disk or other form of neuritis visible, at least to the ophthalmoscope.

Hæmorrhage into the Pial Sheath of the Optic Nerve.—Michel† has reported an interesting and very instructive case of this occurrence which was accompanied with the ophthalmoscopic appearances of neuritis.

The patient was a man of sixty-five, who died rather suddenly with apoplectic symptoms. There was at the time paralysis of the ocular muscles; vision was not, however, essentially affected. The ophthalmoscope showed that there was a moderate degree of hyperæmia of both disks, with marked filling of the veins. Twenty-four hours later there was a decided change in the ophthalmoscopic appearances of the papilla of the left eye. The upper and outer borders of the nerve had faded away and were lost in a grayish-white disturbance of a peculiar brilliancy, which at times seemed to play into a violet shade. The venous vessels were much distended and very tortuous. In another twenty-four hours the same changes had taken place in the other eye.

These symptoms gradually increased until the death of the patient, accompanied by a moderate swelling of the disk. There were uræmic symptoms preceding death.

At the autopsy a cysticercus was found in the right hemisphere of the cerebellum; also a light degree of internal hydrocephalus. There was granular atrophy of both kidneys, without any hypertrophy of the heart and without any lung disease.

The eyes were opened *in situ* by an equatorial section, and both disks were found to be untransparent and indistinct in their outlines. Everything else was normal. The only thing to be seen was a stri-

* "Trans. Ophth. Soc. of the United Kingdom," vol. iv., p. 274.

† Archiv für Ophth., Band xxiii, Ab. 11, p. 213.

ated extravasation which was situated upon the upper surface of the left nerve, about twelve millimetres from the bulb. This hæmorrhage was about two millimetres long and one third of a millimetre broad, and was evidently either in the pial sheath or in the tissue of the nerve just beneath it. A microscopical examination of this part showed that the nervous elements had completely preserved their integrity. There was, however, some infiltration of the connective tissue with lymph corpuscles. The pial sheath and the septa derived from it were infiltrated in a uniform manner, with round cells, as was indeed the nerve tissue, only to a less degree (Fig. 32). There was scarcely any swelling of the disk, but it, as well as the surrounding tissue, was infiltrated with round cells. The bleeding into the pial sheath and its processes is shown in the figure. To the naked eye these hæmorrhages appeared as dark stripes (*b*), and dark points (*c*). With the microscope, these places consisted of red blood-corpuscles closely pressed together. These occurred only in the region of the connective tissue; and in the extensions of the pial sheath they were chiefly found at the intersections of the staging work (*c*). Red blood-corpuscles were in no case found in the nerve tissue itself. The hæmorrhages were not frequent into the pial sheath proper, but were more so in the extensions from it, which form the septa between the nerve bundles, and were distributed at intervals along the whole course of the optic nerve from the chiasma forward.

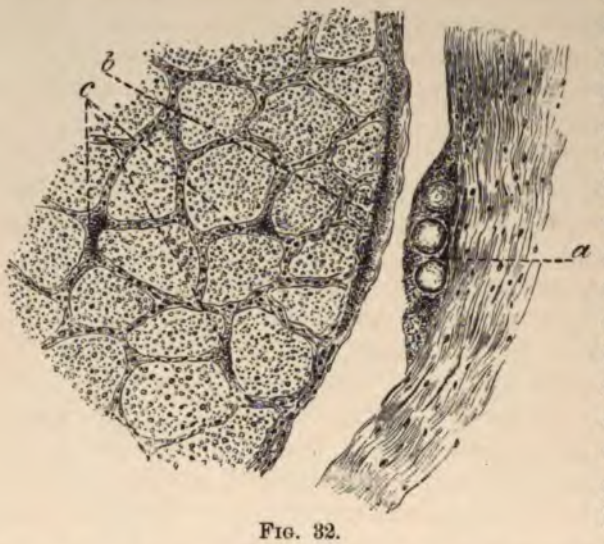

FIG. 32.

Michel looked upon the hæmorrhages as the primary affection which produced disturbances in the nutrition of the tissue, which were the cause of diffuse infiltration, and the presence of the round cells.

Hyperæmia of the Central System.—This may be either an ex-

pression of a general hyperæmia or of a local one which is limited to the eye. This latter may affect both the arteries and veins, and then usually predominates mostly in the latter. In rare cases the opposite may occur, and the arteries be more affected than the veins. It must, however, be very exceptional if it ever occurs that the arteries alone are affected. This kind of local hyperæmia simply signifies that a larger quantity of blood is present in the eye than is usually found in the normal condition, and finds its expression simply in a dilatation of the larger vessels of the retina. Neither the disk nor the background of the eye appears essentially altered, which condition may continue for years, and which, when accompanied by asthenopic symptoms, where there is no error of refraction, often occasions a doubt as to whether the vessels are really abnormally increased in size, or whether it is a congenital peculiarity.

In marked cases of hyperæmia, besides being dilated, the vessels have occasionally an antero-posterior as well as a lateral curvature, which is shown then by alternating light and dark segments of the vessels. On account of dilatation of the finer vessels, many more become visible, not only on the disk, but also in the substance of the retina. In the region of the macula, where only the finer vessels run, the ramifications are sometimes very clearly marked and can be followed up to the fovea.

Capillary Hyperæmia.—The capillary network of the retina is too fine to be recognized as single vessels, but vessels which are originally too small to be seen with the ophthalmoscope often become visible under hyperæmia. Thus, capillary congestion makes itself manifest through increased redness of the disk, and it is only under certain conditions, to be dwelt upon later, that hyperæmia of the retina itself is visible, even with the upright image. In capillary hyperæmia, as a rule, the "light spot" which marks the centre of the disk usually remains, even when the congestion of the rest of the papilla is very marked. It is of service to note this fact, as it forms an important diagnostic mark between the simple dilatation of the finer vessels and the congestion due to some forms of neuritis, where the spot which marks the physiological excavation is the first place to fill up and become congested. There is, furthermore, a form of capillary hyperæmia where the entire papilla is not only reddened in a diffuse manner, but where also numerous small and fine vessels, closely pressed together, run in a radial direction, but which are only visible here and there in their course. These vessels extend into the retina itself, passing over the connective-tissue ring which they then have a tendency to obscure. This is often found in that form of increased

vascularity which may be classed as due to "retinal irritation." This increase of the finer vessels radiating from the disk also forms an important diagnostic mark between the increased amount of blood in the retinal vessels and that of a congestion of the disk which accompanies choroidal disease. In this latter the injection of the nerve is more central and the connective-tissue ring, instead of being obscured, is rendered more conspicuous and more sharply defined by contrast. This effect is often very marked in choroiditis disseminata, and sometimes in the lower forms of choroiditis posterior, such as accompany some stages of progressive myopia.

Stasis Hyperæmia.—This form of hyperæmia results from any cause which interferes with the circulation, so as to impede the entrance of the arterial or the exit of the venous blood. A sharp distinction should be drawn between this form of hyperæmia, which may be called passive, and that which is called forth by an active determination of blood. It is characterized by an increase in the size of the veins, and either a normal condition or a narrowing of the arteries. This increase of the veins may be very great, and be expressed by a large amount of tortuosity of the vessels. The papilla may be reddened to a considerable degree, and sometimes be marked by dilated vessels, which run in a radial direction. In marked contrast to this is the behavior of the arteries which are narrower and run in a straighter course than in the normal condition. This form of hyperæmia takes place in many if not most kinds of retinitis, even in those in which there is no evident sign of impediment to the circulation outside of the eye itself. It is common, under these conditions, to attribute the narrowing of the arteries to compression from swelling of the tissue, especially at the point of passage of the vessels through the non-yielding tissue of the scleral ring opening. It would appear, however, that too much stress had been placed upon this as a cause of retardation of the current, even in what has been denominated as choked disk, as microscopical investigations by several observers—notably by Pagenstecher and Gowers—have failed to show that there were any constrictions of the vessels from outside pressure upon their walls. It is, therefore, in many if not in most cases due to a morbid condition of the walls themselves, which leads to a retardation of the circulation, either from an altered condition of the blood or from some vaso-motor disturbance, which incites a spasmodic contraction of the muscular coats of the arteries while it favors a distention of the veins, on account of a reduced *vis a tergo* from a lessened arterial supply, aided in its turn by the effect of intra-ocular pressure.

It would seem as if the speedy and easy exit of the venous blood

was necessary for the full distention of the arteries. As if a vacuum, for example, created in front of the arteries by the escape of the venous blood, increased the *vis a tergo* from the arteries, and that there took place a sort of siphoning out of the venous, which was replaced by a more rapid flow of the arterial blood through the capillaries.

At any rate, when thrombosis of the central vein has been shown to exist, so that blood could not flow out of the eye, the arteries were smaller than normal, sometimes to a marked degree, so as to give the attenuated appearance due to embolism.

Collateral Hyperæmia.—This is said to occur in embolism or other obstruction to the circulation, in a greater or lesser degree, according as the impediment is in a larger or smaller branch of the central artery, or as the closure is partial or total. I must say that, personally, I have never seen any appearance of distention of the veins after embolism which did not appear to me more compatible with the belief that the wall itself was relaxed and distended from stasis and its softening influences, and that the hæmorrhages were the result of such a condition rather than due to increased pressure within the wall, caused either by reflux through the veins, or from a direct and abnormal fluxion in the arteries.

Atonic Hyperæmia.—This, as it occurs in some forms of retinitis during retrogressive changes, has already been mentioned in a general way, under the heading of "Increase in the Diameter of the Veins" (page 5). It is sometimes very intense in its character, so that the veins are three or four times as large as the arteries. Some veins are at times more distended than at others, and are more distended in one portion than another. Usually the dilatation is most marked in those places where the inflammatory action has been the greatest. Whether this is due to vaso-motor influences, or to a simple yielding of the vessel while in a relaxed condition, is not yet determined. The fact that the dilatation takes place where no disease in the wall can be detected, and that it may be accompanied by arteries which are abnormally contracted, would point to the conclusion that there might be some paresis, through the agency of which the veins become dilated, while there was a contemporaneous vaso-motor irritation and consequent contraction of the arteries.

Neuro-paralytic Hyperæmia.—From the very marked and conspicuous congestion of the vessels which section of the sympathetic nerve in the neck produces, it would naturally be expected that paralysis of the cervical sympathetic would be followed by pronounced changes at the back of the eye, that would be plainly if not con-

spicuously visible with the ophthalmoscope, and it would be supposed that this must take place from anatomical as well as physiological reasons, since it has been, it seems to me, proved beyond question that sympathetic fibres arrive at the eye through various channels, and accompany not only the ophthalmic but also the central artery at least as far as the disk, and probably much farther into the retina itself, following the general law, and attending the ramifications of the branches to which they are distributed, no matter how delicate these become. The same is true of the choroid and the whole uveal tract. Moreover, physiology teaches us that, if we divide the cervical sympathetic, or extirpate the upper cervical ganglion, the circulation in all of the vessels going to that side of the head, both external and internal, is increased; the pia mater becomes injected, as do the ear, the cheek, and the conjunctiva, as well as the iris. It is, moreover, directly asserted by Leber * that the retinal arteries are innervated by the filaments from the cervical sympathetic. He states, however, that section of the nerve produces only a slight dilatation of the arteries, and declares that "whether the slight effect of section of the nerve is to be attributed to the action of the intra-ocular pressure or other causes could not be determined." O. Becker also says that the effect upon the retinal arteries was much less than upon the vessels of the ear.

Klein and Svetlin,† after carefully isolating the sympathetic in the neck in cats, got no effect whatever upon the retinal vessels or those of the optic nerve, either by irritating the divided end of the nerve, or the entire cervical portion, or by extirpation of the ganglion.

Others, it is true, have got what they claim to be decided effects by irritation and division. But it appears to me, after a careful examination of the evidence, that the weight of authority is in favor of the fact that the effects on the circulation of the retina and optic nerve by experimentation on the cervical sympathetic are, though perceptible, very slight, and that the contraction which takes place by irritation is much greater proportionately than the dilatation which follows section or extirpation. Also the disproportion mentioned by Becker between the effects of section of the nerve upon the ear and external parts of the head, and those upon the back of the eye, would seem to hold good even to a greater degree in man than in animals. Thus, Mitchell,‡ Ogle,§ and Samelsohn,‖ have reported cases where the cervical sympathetic was affected, and where all the symptoms of

* Graefe and Saemisch, vol. ii., p. 353.
† "Wien. med. Presse," 1877, No. 4.
‡ "Injuries of Nerves," p. 319.
§ "Medico-Chir. Trans.," vol. lii., p. 151.
‖ "Archiv für Ophth.," Band xxi., Ab. III., p. 69.

vaso-motor paralysis were present, such as flushing of the ear and cheek with increased temperature, injection of the conjunctiva, and contraction of the pupil, and yet no change could be detected with the ophthalmoscope in the retinal vessels or those of the optic nerve. The more modern literature has many other cases of injury and disease of the cervical ganglion, with all the signs of vaso-motor disturbance in the parts dominated by it, and yet with either no observable change in the retinal circulation, or, at the most, only "a slight hyperæmia of the disk"; and what this means we all know, and that it may and often does last a life-time. Not a single case of even the slightest retinitis or neuro-retinitis can I find in all the literature of these cases, let alone a genuine choked disk.

So, too, when artificial vaso-motor paralysis * is produced by nitrite of amyl, not only do the ear and cheek become flushed, but there are sensory symptoms from dilatation of the cerebral vessels, and yet the circulation of the retina, which is a direct offshoot of these very vessels, is comparatively unaffected.

There would seem to be a good reason for this in the different conditions of the vessels of the retina, the arteries of which do not pulsate visibly in the normal condition, which is evidently due to the counterbalancing effect of the intra-ocular pressure, which maintains an exact equilibrium between the increased impulse of the systole of the heart, on the one side, and the contents of the globe on the other; for, if the pressure is reduced by letting off the aqueous humor, then the arteries pulsate as do the other arteries of the body. This is evidently also the reason why the contraction of the vessels is greater under irritation than the dilatation is under extirpation of the ganglion, for the intra-ocular tension would favor contraction, while it would oppose dilatation.

But it may be asserted that, besides the upper cervical ganglion, there is another vaso-motor centre for the retina and nerve. This is very true; there may be another centre. It has never been found, however, and, even if it were, and other sympathetic fibres besides those now known were traced from this centre to the eye, even then it is fair to assume, judging from the very slight effect produced by irritation or paralysis of the fibres we do know about, that a similar want of effect would be produced by the morbid innervation of those of which we know nothing.

My own experience in these cases is limited. I have seen, however, several cases where there were undoubted symptoms of paralysis of the cervical sympathetic. In only two of them were there any

* Wadsworth and others.

ophthalmoscopic appearances which could not be fairly considered as within physiological limits. In the first case, besides the flushing of the ear and face, there was a decided and unusual flushing of the disk. The other case was one shown by Dr. Amidon to the New York Academy of Medicine. It was that of a young married woman who had been operated upon for a tumor of a sarcomatous nature in the cervical region. It was supposed that the fibres of the nerve in this region had been severed during the operation. All the symptoms of sympathetic paralysis were present in a marked degree, including a conspicuous narrowing of the pupil, showing that the vessels of the interior of the eyeball were affected. The changes in the retinal vessels were also marked, but this was confined to the veins which were palpably enlarged but not tortuous. The arteries were not dilated at all, and seemed to have their normal appearance in every respect, nor did the circulation of the disk seem to be altered in any way. The color of the blood in the veins was somewhat darker than usual, which might have been due in some degree to the narrow pupil preventing a sufficient illumination. Only the eye on the same side of the neck as the tumor was affected.

Jaeger also makes the general statement that, in paralysis of the vaso-motor nerves, only the veins are affected, the arteries remaining normal and the disk showing no essential change.

Cyanosis Bulbi.—This curious and very rare condition usually occurs with malformations of the heart. Sometimes the veins alone, and sometimes both veins and arteries, are affected. They show a very marked dilatation, which takes place over the entire body, especially in the smaller vessels of the skin and mucous membranes. The blood in both systems is usually much darker than normal, that in the arteries being as dark as the ordinary venous blood, while that in the veins is of a dark violet-blue.

A beautiful specimen of this condition was reported to the New York Ophthalmological Society by Dr. W. O. Moore, through whose kindness I had the opportunity of making an examination.

The patient was a young woman of twenty-eight. She had never noticed that her skin was darker than that of other people until six years ago, when she was shipwrecked, during which she was much terrified. Her finger-nails were, however, club-shaped, and she had been informed by her mother that this had been the case since she was ten months old. Her skin is now of a livid, bluish tinge, with dark-purple spots upon the face and hands, and she thinks that the color is increasing in darkness. The ears are purplish, and the conjunctiva a good deal injected.

The veins of the fundus are enormously enlarged and tortuous, and have an antero-posterior curve in certain places, but only on the disk and its immediate neighborhood. There are very many vessels that are plainly visible, and which can be traced far into the retina, which, in the normal state, are either not present or are so minute as to be invisible. The color of the blood in the veins is of a violet tinge. The arteries are considerably enlarged, but not so much as the veins. The blood in the arteries is of the color of ordinary venous blood. There is no pulsation in either arteries or veins. An exhaustive examination by an expert failed to bring to light any disease of the heart or any abnormity in its sounds. The ophthalmoscopic picture presented a strong similarity to that of choked disk, and had been, indeed, taken for that.

The usual anatomical malformations present in these cases are stenosis of the pulmonary artery, patency of the foramen ovale, and abnormal origin of the aorta and pulmonary arteries.

Knapp also reports a case where, during life, there were no abnormal murmurs, in which no valvular trouble was found, but only dilatation of the heart and the general vascular system.

It is probable, therefore, that the cause of the trouble does not always depend upon a defined stasis in the venous system, but may sometimes be produced by a general retardation of the current by a congenital dilatation of the entire vascular system; an enlargement which, if originally congenital, may be increased during life, as was evidently what had occurred in the case just reported, in which no change in the color of the skin was noticed until after the age of twenty-two.

Liebreich gives a drawing of this affection in his "Atlas." In this case the veins are alone involved. [See vol. i., p. 264, Plate III., Fig. 2.]

It would also seem that the color of the blood comes, in part at least, from want of oxidization, as well as from a direct mixture at the heart of venous and arterial blood.

Hyperæmia from reduced Intra-ocular Tension.—This has already been spoken of under the heading of "Increase in Diameter of Veins," page 9.

Hæmorrhage from the Retinal Vessels.—Hæmorrhages may take place from any part of the retina. When few in number they usually occur in the neighborhood of the nerve, and near the larger vessels. When the hæmorrhage lies along and immediately in contact with the larger vessels, it is safe to infer that the hæmorrhage came directly from the vessel itself, and not from the capillaries and smaller vessels

derived from it; especially as the clot sometimes lies directly over the vessel in the most anterior portion of the nerve-fibre layer, and especially, too, as Hoffmann gives a drawing in which the hæmorrhage was seen to lie in the lymph space surrounding the vessel.

It would be supposed *a priori* that hæmorrhages would occur most frequently at the periphery of the field where the vessels are more minute, and for the same reason it would be inferred that hæmorrhage from the capillaries would be more frequent than that from the larger vessels. Such, however, is not the case, and it follows that the pressure within the walls of the smaller vessels and capillaries can not be as great in proportion to their power of resistance as it is in the larger vessels. For the reason that although the sum of the outside diameters of the branches of the vessel is greater than that of the parent vessel, the sum of the inside diameters or the lumen is less, which amounts to saying that the walls are thicker, and therefore stronger in proportion to the strain put upon them in the smaller vessels than in the larger.

Moreover, the occurrence of hæmorrhage near the nerve is also favored by the fact that the influence of the intra-ocular pressure is more felt at this place, and a greater impediment to the onward movement of the pulse-wave would be experienced here than elsewhere; consequently, the effect of sudden and violent action of the heart from over-excitement or abnormal conditions, as well as that of sudden straining, such as in vomiting or coughing, would be more apt to be expressed at this point than at the periphery of the field. This would, of course, hold good all the more where the strength of the wall of the vessel had been reduced by morbid processes.

There are certain cases, however, where capillary hæmorrhage predominates, and where the bleeding is more apt to take place at the periphery of the field than near the nerve, and in the outer rather than in the inner layers of the retina. This goes to show that there is some morbid process which has reduced the strength of the walls, and that it is either confined to the capillaries and small vessels or is expressed in them to a greater degree than in the larger ones. This is an important fact to bear in mind, for capillary hæmorrhage, when of any extent, is much more significant and of graver import in regard to general disease than hæmorrhage from the larger vessels, and all the more, the deeper it lies in the substance of the retina.

Next to the nerve, and its immediate neighborhood, the most frequent place for the occurrence of hæmorrhage is the region of the macula, where central vision is often seriously impaired by the presence of even a small clot. When the bleeding is slight here, and spread

out into a thin layer, it is apt to give to the patient the impression of a red tinge.

Just as hæmorrhages may occur in any part of the surface of the retina, so they may take place in, or extend to, any layer. If the hæmorrhage occurs in the nerve-fibre layer, the bleeding extends itself in a linear or radiating direction, especially in those forms of retinitis in which there is much swelling of this layer. If the bleeding is in the middle or outer layers, this tendency to spread out horizontally is wanting, as the blood-corpuscles have an inclination to follow the connective-tissue stage-work. Thus we are enabled to judge pretty accurately by the shape of the hæmorrhage whether it is in the inner or outer layers of the retina.

Extended hæmorrhagic extravasations are often made up of groups of small red points, and where this is the case the surface of the hæmorrhage has a stippled granular appearance, that is very characteristic. Such hæmorrhages are usually situated in the middle and outer layers of the retina, and are probably of capillary origin or from very small vessels. They are sometimes very extensive, and yet may be entirely absorbed in the course of long-continued inflammations, such as retinitis albuminurica and certain forms of neuritis. Thus I have seen cases of several years' standing where there was no œdema into the retina and no hæmorrhages from the larger vessels, and yet where the extravasations in the outer layers were from time to time very extensive, showing that the finer vessels and capillaries were diseased. These extravasations are probably mostly due to diapedesis.

Some of the larger hæmorrhages which occur in the substance of the retina may traverse the membrane to its posterior surface, and there cause a lifting of the retina from the choroid, which can not be told by the mirror, unless of considerable extent. I have, however, seen a case in which the entire retina was separated from the choroid by an enormous gush of blood. At the first sight there seemed to be no reflex at all from the fundus, and I was upon the point of supposing that the vitreous was filled with blood. I obtained a perfectly fair and unobstructed view of the disk. The vessels could only be traced for a short distance beyond the borders of the nerve, and the movements of the retina seemed like the rising and falling of a black wave as it alternately disclosed and shut out the view of the disk.

Contrary to the usual belief, it is very rare that retinal hæmorrhages are transformed into patches of pigment. Even extravasations of considerable size are completely absorbed without leaving much if any textural change, or indeed any sign of their existence, behind. It is only when the bleeding has been very profuse and long continued

that spots of a brownish-black color are found, and even in these cases it usually signifies that there has been some alteration of a cicatricial kind in the tissue of the retina itself. The fresher the hæmorrhage is, the brighter the color, so that old hæmorrhages are usually darker than recent ones, although they very often retain their vivid red color until they are entirely absorbed. This is probably due to some chemical condition of the blood which does not allow it to part with its hæmoglobin. This occurs more frequently in capillary hæmorrhage, and naturally where the hæmorrhage is spread out in a thin layer.

In the reabsorption the red corpuscles are in part directly decomposed, and in part are taken up by the lymph corpuscles, and become changed in their interior into a fatty, glittering substance and pigment, which remains in the tissue in case cicatricial changes have taken place. The white central spots that occur in the middle of the clot consist of granular cells, which are at times colorless or of a yellowish-red, or darker in case the bleeding has been profuse.

Hæmorrhages from the retina into the vitreous are very common, while, contrary to what has been usually supposed, those from the choroid are very rare.

Hæmorrhages from the retinal vessels take place in two ways—either from rupture or from diapedesis. According to some authors, the latter is the most frequent, if not the only, way in which hæmorrhage occurs, except from rupture by concussion, and even here rupture of the retinal vessels with bleeding very rarely takes place unless the enveloping membrane of the eye is broken.

The blood-corpuscles probably pass singly through the walls of the vessels, from the fact that so little injury is done to the retinal tissue. The number of the corpuscles is greatest in the neighborhood of the vessels from which they come, while beyond it they become fewer and more scattered. This would explain the effect seen with the mirror, of striated hæmorrhages which follow along the walls, and the punctate form which the bleeding takes when at a distance from the vessel. The blood-corpuscles show a tendency to accumulate beneath the internal limitans, and at the macula large collections of these occur, so as to form at times extensive hæmorrhagic plaques. Liebreich gives a remarkable picture of one of these extravasations at the macula, which consists of an immense oval plaque, and which occurred from suppression of the menses in a woman of forty-five.* After a little while these hæmorrhages assume, by gravitation downward, a semicircular shape, while the upper segment becomes of a

* "Atlas d'Ophthalmoscopie," Tab. viii., Fig. 2.

grayish color, probably due to a turbid fluid behind the limitans, and to a want of transparency in the membrane itself. White spots also occur, either within the circumference of these hæmorrhages or in their neighborhood, and may assume such a size as to resemble large patches of exudation with a glittering sheen to them. Leber cites several cases of this form of hæmorrhage, from suppressed menstruation, and also states that it may occur in young people of both sexes, and that it is often accompanied or rather preceded by one-sided epistaxis.

I have myself seen one-sided hæmorrhage at the macula lutea in young men for which there seemed to be no adequate cause, and the same fact has been recorded by Eales and others.

Besides occurring in all forms of inflammatory diseases of the retina, hæmorrhage may take place through increased action of the heart from hypertrophy, in fluxions, and disturbances of the circulation and suppressions or irregularities of the menses; in sclerosis and fatty degeneration of the vessels, in miliary aneurisms in the retina, and in thrombosis and embolism; in general diseases where the constitution of the blood is changed, as in maladies of the kidneys and spleen; in diabetes and leukæmia, pernicious anæmia, tuberculosis and gouty affections, as well as those which represent a hæmorrhagic diathesis, such as scrofula and purpura.

Anæmia of the Central Vessels.—Most of the symptoms of anæmia and hyperæmia have already been related somewhat in detail in the general description of the changes in diameter and color of the vessels as they are found in different conditions of the system. Still the matter is so important that the principal symptoms are grouped together here for the purposes of uniformity and for easy reference.

Anæmia of the retinal vessels would appear to possess two chief characteristics—one that relates to the reduced quantity of the blood which the vessels contain, and the other to the composition of the blood itself.

So far as the amount of blood is concerned, the most important symptoms consist of a uniform decrease in the diameter of the vessels usually for their entire course, a lighter color of the blood from a reduced size of the vessel, and a corresponding decrease in the breadth of the light-streak.

The decrease in the diameter of the vessel begins in the perpendicular diameter—that is to say, in an antero-posterior direction, so that the vessel appears a little flatter in the horizontal diameter. The length of the vessel is not essentially altered, nor do the vessels show any essential change in their course or tortuosity. Still, in exag-

gerated cases they do begin to be less visible, or are entirely lost to view toward the periphery from the disk, so that in some cases of feeble heart, on the one side, and disproportionately increased intra-ocular pressure on the other, the arteries have a peculiarly stunted look, as they still maintain their original dimensions near the disk and become rapidly attenuated or lost to sight at a comparatively short distance from the papilla. The proportion between the arteries and veins is generally maintained—that is to say, both arteries and veins are reduced in an equal degree, especially when the reduction in size is the expression of some general toxic disturbance, as in some cases of amblyopia potatorum and tobacco amaurosis, which in their earlier stages before vision has become much, if at all, affected, present the appearance of simple anæmia.

There are, however, exceptions to the rule that the proportion in diameter between the arteries and veins remains the same, for cases often occur, as has been already mentioned, where the arteries alone are reduced in size, and where the veins remain normal or may be increased in size.

It may be remarked here that Becker mentions having sometimes observed a spontaneous arterial pulse in cases of chlorotic girls and in Graves' disease. The color of the vessels is usually only affected in a trifling manner, although the blood becomes lighter in both the veins and arteries as the vessels become smaller, until in the higher degrees the arterial blood is hardly to be differentiated in shade from the yellowish red of the fundus.

The reflex is at first broader as the vessels are flatter. Later it becomes narrower as the vessels become reduced in all their diameters, and it also becomes less brilliant, until it fades entirely away in some cases. In these I am of the opinion that the wall itself has become less transparent, and that the reduction in the size of the vessel is not due to anæmia, but is really caused by a slowly progressing and low type of disease in the walls, no doubt aided by the increased thickness of the wall by its contraction upon itself.

The fundus, as a whole, in anæmia appears somewhat paler than normal, and the outlines of the nerve are a little less sharply defined than usual, because the contrast between the surface of the disk and the surrounding tissue is less marked, but there are extreme cases of anæmia where the capillary circulation is so much reduced that the contrast is more marked, and some care is required then not to confound simple anæmia with beginning atrophy.

Anæmia in the retinal circulation may be the local expression of a general anæmia, which arises from any cause which seriously im-

pairs the bodily functions or the quality of the blood. There is produced under such conditions what may be called a functional ischæmia in the vessel, in contradistinction to the spasmodic variety, which has already been described. This occurs particularly after severe loss of blood from any cause or from any organ, and especially that incident to gestation. This loss of blood may be accompanied by a greater and lasting impairment of vision. The failure of sight in these cases is proportionate to the quantity of the hæmorrhage, the number of the attacks, and the rapidity with which the blood is lost. If the hæmorrhage is not repeated, and is not severe, the recovery is oftentimes very rapid. If it is repeated, successive deteriorations occur, changes in the optic nerve become more apparent, and the vision goes from bad to worse. In such cases there is usually very little change in the fundus beyond a greater or lesser attenuation of the vessels, and in that the capillary circulation fails more than that of the central vessels, which gives a slight bluish tinge to the nerve. Sometimes, however, the changes are not limited to the vessels alone, but affect also the retina, which then becomes the seat of an inflammatory action, that usually shows itself in a mild form of retinitis, with some œdema, in the neighborhood of the disk. Cases, however, have been reported by Hirschberg, and others, where the bleeding was followed by an intense neuro-retinitis, with plaques of exudation and hæmorrhage.

Anæmia in the retinal vessels, as an expression of a general anæmia, is much less common than is supposed, and we often see persons with a pale complexion, or one of marked pallor, where the retinal vessels are not only normally filled but where there is no reduction in color. In such cases we have the right to assume that the color of the blood is the same as in the normal state, but that for some reason the small vessels and capillaries of the skin are reduced in size, and that the paleness is due to the reduction in the contents of the vessel, perhaps of the same nature, but of a more permanent character, as that which occurs in sudden and temporary paleness, and which might come from some vaso-motor disturbance.

This might explain why in some cases no amount of iron tonics increases the redness of the complexion. On the other hand, there are some cases where an examination of the blood shows that there is undoubted anæmia, but yet where the face is not only not pale, but even where it may be of a rosy color. This is no doubt due to the fact that, although there is an anæmic condition of the blood, the superficial vessels and capillaries of the skin are more dilated than common, probably from a retarded circulation, so that while the ophthalmoscope shows that the blood is really lighter than usual, and

hence that iron tonics would be of service, the complexion would not show it, but even contra-indicate its use.

Simple Anæmia.—Variations in the composition of the blood often produce changes which are readily detected with the ophthalmoscope. The principal change is naturally that which relates to variations in the color of the blood, and this change is not one that depends upon the greater or lesser diameter of the vessels, but one which is caused by an actual change in the composition of the blood and in the proportions which its elements bear to each other. Thus, in simple anæmia, where the hæmoglobin is lessened either because the red corpuscles are reduced in quantity or in size, or in richness of the coloring matter, the blood in the retinal vessels is paler than normal, as is also the general fundus. This usually takes place in anæmia in both arteries and veins, and the same holds good in hydræmia, or its equivalent, hypalbuminosis, where the solid contents of the blood are reduced in comparison with the watery elements. Thus, in Bright's disease, where hypalbuminosis is most marked, the blood in the arteries, especially before retinitis has ensued, is sometimes of an exceedingly light color, with a broad and glittering reflex, while the blood in the veins is also much lighter than normal. But this is not always the case, for when the retina has become implicated, and especially when hæmorrhages have occurred, then the blood in the veins becomes of a darker color, so that there may be at the same time an abnormally dark venous and an abnormally light arterial blood, with narrower arteries and broader veins.

Not only is the color of the blood affected by the proportion of its elements, but other changes of a marked and interesting character follow the alteration in its composition. Thus, it has been shown that anæmic and hydræmic blood have a peculiar effect upon the condition of the walls of the vessel, so that hæmorrhages from diapedesis and exudations take place through the capillaries and small vessels, which are sometimes of considerable degree, as seen by the ophthalmoscope; so that in some cases of excessive anæmia we see tissue changes in the fundus, such as are expressed not only by extravasations but also by all the inflammatory signs which are characteristic of neuritis, neuro-retinitis, and choked disk of the most flagrant form.

From a careful study and tabulation of the richness of the blood, as to the number of corpuscles, and the quantity of the hæmoglobin present, Dr. Stephen Mackenzie has come to the conclusion that when the corpuscular richness falls below fifty per cent of the normal, the tendency to hæmorrhage becomes developed, while the quantity of

hæmoglobin exercises a qualifying influence, according as it is greater, equal, or less than the corpuscular deficiency.

Pernicious Anæmia.—In cases of that extreme degree of anæmia to which the name of idiopathic or progressive anæmia has been applied, retinal hæmorrhage is very common, and all but constant. In such cases the corpuscular richness falls to as low as twenty, fifteen, or even ten per cent.*

The retinitis that accompanies pernicious anæmia is usually mild in form, and not as a rule accompanied by signs of active inflammation. There is usually very little swelling of the tissue, or œdema, and the nerve is not implicated to any great degree, though there may be, and usually is, combined with the retinitis a low stage of neuritis which shows itself by a slight obscuration of the edges of the disk, and a delicate striated or woolly appearance of the papilla as it passes over into the retina.

The most conspicuous change, and the one that is first noticed as a rule, is the light, yellowish-pink shade which predominates over the background of the eye. It is similar to that which is found in the graver forms of simple anæmia, only more marked. This is evidently due to the blood itself, and not to a lessening in diameter of the blood-column. The color in both arteries and veins seems lighter than normal, and there is but little difference in shade between the two sets of vessels. The light-streak is unusually broad, and not as a rule very brilliant, although at times it may be so, depending upon the translucency of the wall. The veins are ordinarily somewhat enlarged, and at times, but rarely, a marked tortuosity may be observed. The arteries are, as a rule, as in all retinal inflammations, somewhat reduced in their diameter.

Another prominent characteristic of pernicious anæmia is the tendency toward hæmorrhage. These are usually striated in form, and not round, as in septic retinitis. They lie near the vessels, and usually in the inner layers of the retina. They are as a rule of small size, and flake-like, and for this reason may be rapidly absorbed. They have at times a whitish center.

Although it can not be denied that there is in this form of anæmia a marked tendency to hæmorrhage, it is not so constant a symptom as commonly believed. Indeed, in one of the most marked examples of the disease that I have seen there was no bleeding at all. The case was that of a middle-aged man, in whose blood the red corpuscles were reduced to twenty per cent of the normal amount, and the hæmoglobin to eleven per cent. There was intense pallor of

* "Trans. Oph. Soc. of the United Kingdom," vol. iv., 1884, p. 132.

the face and skin, which resembled white wax in appearance. The fundus of each eye was exceedingly pale also, and there was little or no capillary circulation visible upon the disk, which was of a creamy or yellowish-white shade, but it had none of the characteristic appearances of atrophy. The edges of the disk were not obscured, and there was not a trace of œdema or hæmorrhage round its circumference, or, indeed, in any part of the retina; nor were there any signs of inflammatory action in any of the tissues or membranes. The nerve and the fundus looked as if they had simply faded out to a great degree. The blood in the veins was lighter than ordinary arterial blood, being of a pale-pink color, and that in the arteries near the disk appeared lighter still—almost, in fact, white—though a slight discrimination between arterial and venous blood still remained. The most striking appearance, however, was the condition of the larger vessels upon and near the papilla. The arteries had almost a dead white appearance, not a glittering, or on the other hand, an opaque aspect. Moreover, a translucent effect was produced under the play of light from the mirror, which was very peculiar and difficult to describe. The light-streak was exceedingly broad and very brilliant, appearing under rotation of the instrument almost to flash. It was supposed at the time that this peculiar appearance of the vessels could not be due to the blood alone, but must be caused, in part at least, by the conditions of the wall itself, which was supposed to be the seat of fatty or fibrinous infiltration.

This opinion was apparently sustained by the fact that, as one ran the eye along the main branches of the arteries toward the periphery of the field, where the walls are thinner, the white, translucent appearance of the vessels grew gradually less. Moreover, under pressure, the trunks of the larger vessels remained visible, as fine and rather brilliant threads. This opinion that the walls were affected must either have been incorrect, or a change had taken place in the eyes as a post-mortem alteration.

The autopsy in this case presented in a marked degree all the symptoms which are supposed to be characteristic of the disease that is now known as pernicious anæmia. The eyes were given to Dr. Lennox, Curator of the New York Eye Infirmary, who reported as the result of his examination that "there was a certain amount of dimness of the tissues, especially that of the retina. The nervous elements, apart from this, were apparently normal. There seemed to be no increase of connective tissue, and no hæmorrhages. The vessels, both arteries and veins, were normal, showing neither thickening nor degeneration, but they contained little, if any, blood—indeed, this

absence of blood, even from the capillaries, was the most striking thing about the preparation; no red blood-cells at all could be found in either retina, choroid, or in the ciliary vessels around the optic vein. In some of the smaller vessels there were a few nuclei adhering to the vascular walls."

This agrees with other examinations that show that the structure of the vessel is not usually changed, although the capillaries are distended. Manz found these filled with colorless cells, which would account for the general paleness of the fundus; and I can only account for the peculiar ophthalmoscopic appearances of the larger vessels by the supposition that the plasmic current which surrounds the inner or red blood-column, was so filled with white corpuscles as to reflect a large amount of light, which partially, or where they were the thickest, entirely concealed the inner or red portion of the current.

Similar ophthalmoscopic appearances with those described as occurring in pernicious anæmia are also observed in other diseases in which there is a hæmorrhagic tendency, such as scorbutic anæmia, purpura, scurvy, and malarial fever.

Atrophy of the Retinal Vessels.—This shows itself in a uniform reduction in the breadth and length of the vessels; also in a lighter color of the blood, and in a reduction in the breadth and brilliancy of the light-streak; in the increased visibility of the wall of the vessel, combined with the appearances of retrogressive changes of the rest of the tissue of the retina, and by the appearance of a white or grayish color of the head of the nerve.

It will be seen at once that all these symptoms bear a striking resemblance, in enumeration at least, to those of anæmia. Still, with most of them to a practiced eye there is a decided difference in degree, if not in character, which distinguishes them often at a glance. Thus the contours of the vessel in anæmia present their normal sharpness, and the walls have a pellucid expression to them, which is wanting when atrophy is present, or even beginning. Moreover, the vessels in atrophy become straighter in their course, as if they contracted upon themselves as they became narrower, which is not the case, or at least not nearly to the same degree, in anæmia. This is more noticeable in the veins than in the arteries, as they have a more tortuous course.

The reduction in size of the vessel usually affects both the veins and arteries in an equal degree, so that the proportion between the two is the same, the veins remaining visible for a longer time because they are larger and darker in color; but occasionally I have thought

that the abnormal processes which caused the shrinkage of the vessels were confined to the arteries alone, since the veins either retained their normal size or were larger, while it was certain, from the color of the nerve, that atrophy was beginning. So, too, when the atrophy is secondary to violent inflammation of the retina, the arteries are usually disproportionately small in comparison with the veins, as they always are after embolism or thrombosis.

In the same degree as the vessel becomes smaller, so does the blood-column assume a lighter color, so that the arteries may become very difficult to trace, or invisible on the yellowish-red background, while the veins are still apparent as dark-red lines. When, however, degenerative changes, such as sclerosis, are taking place, which somewhat interfere with the perfect transparency of the wall, the color in the arteries seems to be maintained, or at least their visibility is, as they can often be followed while much attenuated, far toward the periphery. In such cases the arteries seem to become narrower in a greater degree toward the nerve, perhaps because they are thicker. They lose, therefore, their natural tapering appearance, which is one of the earliest signs, to a careful observer, of some change taking place in the walls of the vessel.

As the vessel becomes smaller, the reflex becomes narrower, and if the atrophy is carried to a considerable degree, it loses continually in breadth and intensity, and finally disappears, so that it is completely lost, in some cases of atrophy where the vessels are of such a size that it would be clearly seen in a physiological condition. This shows that the translucency of the wall is being reduced, and that sclerosis or degenerative changes are taking place in it, which may go to such an extent that the wall itself becomes visible. This increased visibility of the walls, and the effects of inflammatory changes upon them, have already been treated of under perivasculitis, to which the reader is referred for a more detailed account.

One of the most distinctive points in the discrimination between anæmia and atrophy is the appearance of the disk, and especially in regard to its color, or what may be termed its complexion. The paleness of the disk in anæmia is simply a want of color, due to an insufficient quantity of blood in the smaller vessels of the papilla, which then has a soft, creamy look, while the pallor of atrophy of the vessels is more conspicuous, of a more brilliant tone, with a bluish-white tinge to it, like that of skimmed milk rather than that of cream; or even at times it may have a tendinous or mother-of-pearl-like aspect. In this case atrophy of the vessels is usually combined with an altered condition of the nerve-fibers, and an increased amount

of connective tissue. At times there is instead of the bluish-white color a grayish tinge, which may be concentrated near the outer border of the nerve in a crescentic shape, which in the very initial stage of atrophy gives rather the impression of an increased than a reduced vascularity.

Another important distinction between atrophy and anæmia is that even the lightest degrees of the former are accompanied, though not always in their incipiency, by disturbances of vision and a reduction of the field of vision.

Atrophy of the vessels can go to such a degree that absorption of certain branches takes place, followed in some cases by that of the trunks; and to such an extent can this be carried that I have seen a case where the entire arterial system had been absorbed, the only vessel which was left being a portion of one of the lower veins at the disk.

Leber is of the opinion that in these rare cases of congenital atrophy of the optic nerve, where there are no vessels at all to be seen, it is rather to be attributed to complete atrophy of the vessels than to a congenital want of them.

CHAPTER II.

IRRITATION OF THE RETINA.

Midway between simple hyperæmia of the retinal vessels and actual inflammation stands a form of congestion that has been aptly designated by Jaeger as irritation of the retina. This form of congestion, although chiefly distinguished from hyperæmia by the presence of functional disturbances, has yet some physical characteristics and features of its own which it is important to notice, especially in these days when all symptoms of asthenopia are referred to an error of refraction, should by chance any such exist, even of an infinitesimal degree.

The congestion due to irritation shows itself principally upon the disk itself, and those parts of the retina which immediately surround it. It lies in the most advanced portion of the head of the nerve, and it is therefore anterior to the connective-tissue ring and choroidal border of the nerve, both of which it has a tendency either to obscure or conceal when the congestion is of a pronounced degree.

The increased redness, taken as a whole, is at the upper, inner, and lower portion of the nerve—that is to say, the nasal portion of the disk. The injection follows principally the course of the larger vessels, and has a radiating or striped appearance, which is the more marked the more abundant are the connective-tissue elements. It follows also the striation of the nerve-fibers, and an appearance of irritation is thus given to the nasal half of the disk, where the fibers exist in greater numbers, which other parts lack, while at the same time the redness is of a more uniform character, and appears to be produced by the smaller vessels. The injected portion has a somewhat sharply defined border toward the porus opticus, and becomes gradually less intense toward the periphery of the disk. As a rule the "light-spot" in the center of the nerve remains unaffected, probably from the fact that the connective-tissue string anterior to the sclera, and consequently in the plane of the lamina cribrosa, contains no vessels except the central artery and vein (see Vol. I., p. 29). There are, however, exceptions to this rule; and then the central part

of the nerve, or that immediately surrounding the vessels, is very much injected. But such cases as these are not cases of simple irritation, but of actual disease. It happens most frequently in mild forms of neuritis and choroidal troubles, and then the increased redness of the "light-spot" forms an important diagnostic mark between retinal and choroidal or scleral disease.

So far as the neighboring tissue of the retina itself is concerned, we can detect by careful observation two kinds of redness: one, which presents a more or less diffuse tinge, and which lies principally in the deeper layers of the retina, and which is probably due to the congestion of the network of capillaries and finer vessels that are situated in these layers (vol. i, p. 35); secondly, a superficial redness that follows the striping of the nerve-fibres, and which is the more pronounced the more abundant the connective-tissue elements are. This increased redness in the substance of the retina extends for a distance of one or two diameters of the disk out into the retina, except in actual inflammation, where it may extend much farther toward the periphery.

In the higher degrees of irritation, especially in persons of dark complexion, the fundus assumes a redder appearance, and the nerve and surrounding region have an angry aspect, particularly when the connective-tissue string becomes congested, and the centre of the nerve reddened. On the other hand, especially with blonde persons, and especially, too, when the irritation has extended to the choroidal circulation, there is a paler look to the fundus, a sort of out-of-focus appearance, which suggests the haze of an Indian-summer day, in which everything is clearly seen and nothing sharply defined—an appearance which is very peculiar and very characteristic when once recognized. This effect is sometimes combined with what looks like a mild form of maceration of the choroidal pigment, which shows itself in streaks and specks of displaced pigment. This kind of fundus is often met with in those forms of asthenopia in which there is either no error of refraction, or one so slight as to be out of all proportion to the amount of suffering.

In the higher degrees of irritation the borders of the nerve are obscured or entirely obliterated, especially at the upper and lower portions, and the fine and striated redness seems then to start from the exit of the central vessels, and to radiate out into the retina for the distance of one or two diameters of the disk, where it gradually passes into a diffuse redness toward the periphery. Under these circumstances the lamina cribrosa loses its reticulated appearance, and the whole look of the nerve is that of beginning neuritis—a con-

dition which may, however, continue for years without any further developments.

The larger vessels do not appear to be affected, as a rule, not being increased either in size, direction, or tortuosity. The process would seem to be limited, like so many other irritations, to the finer vessels and capillaries—to such a degree, indeed, as to suggest the idea that the capillary circulation had an independent power of both expansion and contraction, that was brought about by some force which did not necessarily affect the walls of the larger vessels at the same time. The region of the macula lutea is also in the higher degrees of irritation, more marked in tone, and possesses a deeper color, which may even be combined with the delicate, hazy appearance alluded to in the foregoing passage.

The vision is not affected at all, but one of the most characteristic signs of irritations of the retina, and one which distinguishes it from simple hyperæmia, is the limitation that it places upon the proper functions of the eye. In the well-marked cases there are symptoms which simulate those of asthenopia from errors of refraction, such as fatigue, accompanied at longer or shorter intervals with blur, pain over the brow, and on the temples; a quivering and unsteadiness of small objects, which forces the patient to turn away his gaze; and there may be even a sense of dizziness and nausea, with a marked dread of light.

I have repeatedly seen cases where there was no error of refraction whatever, where the above symptoms were present, and where no other reasonable conclusion could be arrived at but that there was a marked disturbance of function that was occasioned, not by any optical error, but by some derangement of the sensory apparatus—a condition which, like all other neurotic affections, is easy to acquire and very difficult, sometimes almost impossible, to get rid of. In such cases complete and enforced rest, moderate and systematic use of tonics, glasses, and electricity, partial or complete tenotomy, all tried in turn, seem oftentimes to be without avail.

I am inclined to think, moreover, that what subsequently proves to be a serious affection of the eyes, to wit—nerve atrophy—is often preceded by a condition of irritation of the optic nerve and retina. Thus, I have frequently seen patients who complained of a blurring of the type and small objects, after close application of the eyes, where there was no error of refraction, but where a careful examination showed that there was a suspicious whiteness, or perhaps a grayish discoloration of the optic nerve. Or where there was on one side the signs of increased congestion, due to retinal irritation, and

in the other eye the commencing traces of discoloration, due to atrophy.

According to Jaeger, a further exciting cause for irritations of the retina arises in the development and course of various abnormal processes which, under symptoms of irritation or inflammation, occur in more or less distant organs, such as violent inflammations of the conjunctiva and the mucous membranes of the nasal passages, of the larynx, trachea, and bronchi; troubles of the lungs, inflammation of the brain and its meninges; also, as the after-results of measles, scarlet fever, lying-in, and other exhausting diseases.

The fact that troubles in the nasal passages give rise to functional disability of the eyes has lately received a new impetus from the observations and success of the treatment of various practitioners, who, with good sense, have turned their attention from the prescribing of glasses, with almost imperceptible degrees of curvature, to the medication of the real seat of the disease.

CHAPTER III.

INFLAMMATION OF THE RETINA.

WHATEVER it may be to pathology, it is certainly a matter of convenience to recognize, so far as the ophthalmoscope is concerned, two kinds of inflammation — a sthenic and an asthenic form; all the more so, as it usually happens that the sthenic form has its seat, or at least its origin, in the inner layers of the retina, where the larger vessels lie, while the asthenic form takes its rise from the outer, and proceeds toward the inner layers.

It will be noticed, moreover, that in the sthenic forms, where the ophthalmoscopic picture is more pronounced, and the changes much more conspicuous as a rule, the vision is less liable to be affected, and sometimes, even with the most flagrant signs of inflammation, remains perfectly normal; while in the asthenic forms, or those affecting the outer layers, the ophthalmoscopic signs may be very trivial in degree, and yet vision be exceedingly bad. It is certainly marvelous to contemplate how indifferent the nerve-fibres may be to the effects of violent inflammation, by which their very form as well as nature would seem to be changed, as in some cases of choked disk; while, on the other hand, they are so susceptible to the slightest touch of atrophy.

The more sthenic forms of retinal inflammation are characterized by an increased congestion of the vessels which, in the case of the larger ones, is denoted by a marked and unequal enlargement, a darker color of the blood in the veins, with an increased tortuosity; the arteries, on the other hand, are narrower and run a straighter course than even under normal conditions, while their color remains either the same as in health, or, as in some long-standing and debilitating diseases, it becomes lighter than normal.

The injection of the smaller vessels in these sthenic forms of inflammation shows itself in the diffuse and striated redness already mentioned, as occurring in irritation of the retina, only to a greater degree, so that not only is the redness more pronounced upon the papilla and its immediate neighborhood, but also extends far into the retina, and sometimes nearly over its entire surface.

This red color, which is the result of inflammation of the retina, is partly due to the increased size of the vessels and those of new formation, and partly to the extravasation of the red blood-corpuscles. Strange to say, that extravasation occurs less frequently in pure inflammation of the retina than would be supposed; and it would appear to stand less in proportion to the intensity of the inflammation than to a condition of the walls, which favors diapedesis. Thus, in the condition known as retinitis hæmorrhagica, the process is probably much more due to a diseased condition of the walls, or an altered condition of the circulating fluid, than to any inflammatory action going on in the tissue of the retina, since we not infrequently see extravasations of considerable or even great extent, without any signs of active inflammation, or of any loss of function or visual power, except when the extravasations are near the macula; while, on the other hand, we sometimes see all the signs of inflammatory processes of considerable gravity, with a large amount of exudation and no hæmorrhage.

From these changes the contours of the nerve and connective-tissue ring fade away—completely so when the inflammation is of a marked degree. In simple retinitis, uncombined with neuritis, the porus still remains visible, although even this may be entirely obscured in the more violent forms, when the entire fundus is more or less uniformly discolored, and the position of the nerve no longer to be recognized except by the convergence of the vessels (Fig. 33, which is from Jaeger's "Hand-Atlas," Taf. xiii, Fig. 64). The general fundus becomes of a more vivid red color, while at the same time it loses much of its natural brilliancy, and gives the impression of a lack of illumination, or, in other words, as if the light which was thrown into the eye was consumed by the injected and more or less boggy tissue. The outer layers of the retina then become obscured, and the vivid

Fig. 33.

red passes into a grayish red, then into a grayish white, or almost at times a real white, and in such conditions the disk loses its capillary circulation, which is obscured by œdema.

To these symptoms which pertain to the vascular supply may be added also swelling of the retinal tissue, and that of the disk, œdema, exudation, and hæmorrhage.

The swelling of the retinal tissue shows itself, as a rule, in the greatest degree in the neighborhood of the disk, where it is sometimes enormously developed, less at the central parts of the retina, and least of all at the periphery, following thereby the gradual decrease in thickness of the normal tissue. When the swelling is of a low degree, it is difficult to detect it. In higher degrees it shows itself very plainly by the considerable projection of the head of the nerve and surrounding tissue, and by the peculiar tortuosity of the vessels, and their variations in color, the former of which shows itself by a lateral or antero-posterior deviation, which is in proportion to the increased extent of surface of the swollen tissue.

The increase in thickness of the retina is due in a moderate degree to hyperæmia of the vessels, but principally to fibrinous exudations, hæmorrhages, and hypertrophy of connective tissue; also to hypertrophy of the nerve-fibres, which shows itself, as will be seen later, by spindle-formed varicosities.

In considering the effect which swelling of the retinal tissue produces, a distinction must be drawn between a simple lengthening of a vessel and an antero-posterior or twisted course of the same. The first expresses itself, as a rule, simply as a lateral deviation of the vessel, and takes place in the plane of the surface of the retina, and where there is no or very little swelling of the tissue. This lateral deviation, in some forms of retinitis—especially in splenic retinitis, where the vessels, particularly the veins, may curve about and even double upon themselves—is so great that the wonder is that such an amount of circulatory fluid, with a consequent increase of the cubical contents of the vessels, could exist with so little disturbance to the functions of the eye.

Where, on the contrary, there is considerable swelling of the tissue, and especially where this is unevenly distributed, the vessels take on an antero-posterior curve until their direction is sometimes at right angles to the plane of the retina. This is shown by a change of color at the bend of the vessel, while the reflex on this part of the vessel is interrupted—that is to say, on the declivity of the bend, while on the crest it is present in its usual dimensions and brilliancy, unless there is some opacity of the tissue. The deeper the vessel lies in the

swollen tissue, the darker it looks; so that where there is an antero-posterior curve, the vessels appear not only more tortuous but of different shades at different places in the same vessel. This applies particularly to the veins, but it is also applicable in a peculiar manner to the arteries in which the color often appears changed. This is, however, not due to any change in the blood itself, but usually to changes in the surrounding tissue—especially to a lack of transparency; when the outer layers of the retina have a deeper red color from increased congestion of the smaller vessels and capillaries, the arteries, from contrast, appear of a lighter color, as they certainly do where there is an increased amount of fluid in the perivascular spaces.

When, on the other hand, the inflammatory action is principally in the inner layers—that is to say, near the arteries themselves—they usually take on a deeper shade. When the congestion of the retina is carried to a higher degree, and the surrounding tissue is much affected, the difference in color between the column of blood in the arteries and its surroundings becomes lessened—sometimes to such a degree that it is only with great difficulty that the vessel can be differentiated from the rest of the eye-ground, and its course can only be followed by the reflex. Under these conditions the more delicate vessels can not be seen at all, or only during their most superficial course and in isolated places where the changes in the tissue are of a more moderate degree. In this case the retina appears as if poorly provided with vessels, and these, too, are only of the larger kind, being principally veins, which at times appear varicose, or at least wider in some parts than in others, or as if they were interrupted in places and empty of blood. Thus, cases not infrequently occur in the more pronounced forms of retinitis due to Bright's disease and tumor of the brain where, to the inexperienced observer, it seems as if there was actually no arterial circulation at all, and he is often surprised to see on the subsidence of the œdema and swelling the arteries and other vessels come again into view. These, indeed, are sometimes of their original size and direction, and become again almost thread-like in their dimensions from secondary contraction, and atrophy.

As an effect of the inflamed tissue of the retina the underlying choroid loses its firm and clear-cut look, and the whole fundus has usually a more or less smoky aspect, at least in the higher grades of retinitis.

Exudations into the retina in broad and general terms may be classed as serous, fibrinous or albuminous, and purulent. These are characterized by a peculiarity of color under the ophthalmoscope

which in most cases seems to establish their nature. Thus the serous infiltrations have a grayish aspect, which is sometimes of so light a color as to appear almost white, which is then probably due to some change in the nerve-fibre layer, or to an admixture of lymph-corpuscles. It has been shown that the pronounced white color of the vessels, by which they appear to be transformed into untransparent white cords in perivasculitis, is due to the concentric layers of lymph-corpuscles, and it is fair to assume that they produce a similar effect in other places, although infiltration of lymph-corpuscles into the tissue is not so abundant or so frequent in retinitis and neuro-retinitis as is commonly supposed. The fibrinous and albuminous exudations have a whitish-yellow aspect, as, indeed, does that dependent upon fatty degeneration of the tissue; but the former has a softer appearance and a less brilliant aspect than the latter, which has sometimes even a glittering look. These exudations may pass into a grayish white stage, and finally give all the appearances of a membranous nature, containing a large amount of connective tissue.

Purulent infiltration into the retina is very rare, or at least very rarely seen, as, in the diseases under which it occurs, the vitreous humor is commonly so clouded that the fundus is completely concealed from view, as happens almost invariably in what was formerly called metastatic choroiditis or panophthalmitis, as it occurs in parturient women, in cerebro-spinal meningitis, pyæmia, and other grave forms of constitutional trouble. Still, there is a form of genuine septic retinitis in which purulent infiltration can be detected in the retina itself, while the media remain clear. This form of retinitis is usually characterized by a collection of whitish spots of small size, that are usually situated at the macula or the region of the disk.

I am inclined to think that, as the ophthalmoscope is gradually used more and more in general medicine, pyæmic conditions of the system will not infrequently show themselves in the eye by signs and symptoms which will be more fully described under septic retinitis.

In some cases, retinal infiltrations or exudations are so light in color, and, for some reason not yet fully understood, reflect the light so strongly, that they have a great resemblance to plaques of choroidal exudation, but are distinguishable from them with a little care, as they always have a subdued and woolly appearance, with ill-defined edges and a sense of depth which is absent in choroidal exudations. If such changes develop in the outer layers of the retina and optic nerve, then the radiation of the optic fibres has a decidedly reddish, glossy, and striped appearance. The disk reflects more and more light, and then a glassy lustre gradually develops

over the surface of the retina and disk, which is not infrequently met with, especially along the course of the larger vessels, in certain forms of neuro-retinitis from basilar meningitis.

During the development of the changes incident to retinitis, pulsation can readily be produced, as a rule, in both arteries and veins. The arterial pulse can be produced in this case easier even than in health, sometimes even in the most violent forms of inflammation and in the greatest degree of swelling, as, for example, in choked disk, where it is more rapid and forcible, and extends over a greater surface, than in the normal condition. The amount of pressure varies at times, occasionally requiring some force to produce it, while in others it may ensue upon the slightest touch, to stop again when the pressure is increased; and for this reason it is often overlooked. The pulsation in these cases is not usually upon a single vessel, but upon all the arteries up to the edge of the nerve, and even beyond into the retina.

The venous pulse, on the contrary, is usually somewhat harder to produce, is less intense, and extends over a less surface, than in the normal eye.

It is often very important for the observer to be able to determine whether the ophthalmoscopic appearances in a given case are due to a progressive or retrogressive stage of inflammation. This is particularly important in regard to a prognosis, for decrease of vision, always a serious symptom, is more so when the disease follows the subsidence of an inflammation than when it accompanies its rise. When retrogressive metamorphosis is going on, essential changes in the color and appearance of the fundus take place. The congested red color in the neighborhood of the nerve becomes less vivid, and of a darker and more sombre hue. When the changes have extended to the deeper layers of the retina, there is a decidedly grayish, bluish, or even greenish discoloration of the head of the nerve, and of the nerve-fibre layer, which is most clearly expressed in the neighborhood of the disk. I have seen this greenish color so well marked on the borders of spots of old exudations as to resemble a pure emerald-green of a light but very brilliant hue.

When the above changes in color are met with, it is a sign that the active stage of the inflammation has passed, or that the eye has been the seat of a similar inflammation at some former time.

Changes also occur in the vessels. In the first stage of the subsidence of the inflammation the arteries appear to be normally full, or even a little fuller than usual. When secondary contraction begins to take place, from previous implication of the wall, the vessels

begin to grow smaller, and if the action has been severe they may become reduced to thread-like dimensions, either accompanied or not by white streaks along their sides, and other signs of secondary changes.

The hæmorrhages lose their brilliant red and become of a darker color, especially near the edges, which darker color is sometimes followed by a complete de-hæmatization; sometimes, however the blood is absorbed without any change of color, especially where the absorption is rapid, and the clot very small in extent, or very thin and scale-like. On the other hand, in certain forms of retinitis, particularly septic and splenic retinitis, or retinitis albuminurica, the de-hæmatization may take place first in the centre of the clot, so that this appears like a whitish or yellowish exudation with a red ring around it. Should pigment be present in any amount, either in small or large masses, the chances are that the inflammation is retrogressive, or even of an older date than the present attack, as pigment from some simple hæmorrhage is rare, and its presence in the retina generally shows an older process, with cicatricial contraction of the tissue. This becomes almost a certainty when the coloring-matter is found along the edges or upon the surface of membranous expansions, which have a white or glossy look.

Another important diagnostic mark in retinitis is the state of the patient's vision. In simple hyperæmia, even of a pronounced degree, there is usually no disturbance in the quantity of the vision; but in retinitis, even of the milder forms, vision is usually—not always, however—affected, so that there is either a decrease of the amount of central vision, or limitation in the field, of the nature of scotoma or distortion of vision. In this case objects are either reduced in size (micropsia) or altered in shape, especially as to their boundaries (metamorphopsia). This usually manifests itself most clearly to the patient when he is looking at objects which have straight perpendicular boundaries, such as the jambs of doors and the uprights of window-sashes, or, better still, when he is observing a set of parallel lines drawn rather close to each other; such lines then appear to undergo a deviation from their parallel course.

If a contraction or crowding together of the retinal elements has taken place, the lines appear bent outward, as shown in Fig. 34, A. If, however, the elements are drawn apart, then the lines bend inward, as in Fig. 34, B. Sometimes the field has a reticulated character, as if the patient were looking through a sort of grating.

There is oftentimes, too, in many forms of retinitis, torpor of the retina, in which the vision fails disproportionately to a reduction

in the illumination. When, therefore, there is a lack of definite ophthalmoscopic appearances, a careful study of the amount and field of vision will often put the pains-taking observer upon the right track, where a less careful one would have thought the fundus still within physiological limits.

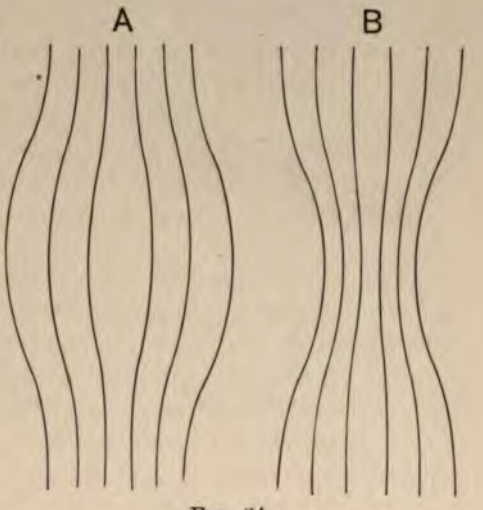

FIG. 34.

Such being some of the more prominent of the general characteristics of retinal inflammation, it remains to notice those forms which, from special features and distinctive qualities have acquired a definite designation. In doing this it will be well for the student to remember that we recognize the individual members of a type or species rather from their differences than their similarities. To the European, so strong is the general likeness of the Chinese face as to make it difficult for him to distinguish the particular individual by his features alone, until, by constant practice, one gets to observe, unconsciously it is true, the *variations* from the general form. It is precisely the same with the features of disease; and he who learns earliest and best to discard similarities, and to note and classify differences, however minute, will make the best diagnostician, not only in ophthalmoscopy, but in every branch of medicine.

CHAPTER IV.

DIFFUSE RETINITIS.

Diffuse Retinitis.—This may arise from any cause, either constitutional or local, and simply means that the disturbance in the retina has extended in a rather uniform manner, and to a greater or less degree, over the surface of the membrane. It occurs usually in the inner layers of the retina, but sometimes also in the outer. In each of these localities it is characterized by symptoms which it is important to notice.

When situated in the inner portions of the retina it attacks principally the nerve-fibre layer, and that immediatelly beneath it, and it manifests itself by a grayish disturbance in the retina, which may vary from a veil-like want of transparency to a thick and dense opacity of a grayish hue which obscures and often conceals the fundus below. The arteries, as a rule, do not appear to be much affected, if at all, and they show very little, if any, increase in size or tortuosity. On the contrary, they often seem reduced in size, and straightened in their course. The veins, on the other hand, here, as in other inflammatory processes, appear increased in size and more tortuous, and have at times an antero-posterior curve to them, as they dip down into the tissue, which they never, or almost never, show in simple hyperæmia. They have also that disjointed appearance, with alternate differences in shade, which is shown when they are more or less concealed by overlying tissue of an opaque nature. By this they are sometimes so much concealed, or completely buried, that the deeper-lying veins are entirely invisible, and only the arteries are perceptible here and there upon the grayish surface of the retina. The veins, however, do not present the same degree of tortuosity as when the nerve is also implicated—that is to say, as we ordinarily find them in neuro-retinitis ; nor are the arteries of such narrow dimensions. Indeed, there are some cases of diffuse retinitis of a mild form in which, to a careless observer, there seems to be little or no change in the vessels, especially as it is only occasionally that fine white lines

are seen to run along the borders of either the veins or arteries, although this sometimes does occur in the severer forms.

In the higher degrees of the inflammation the whole fundus loses its yellow-red color, which gives place to a general grayish or yellowish-gray hue.

The disk is not visible as such, as the innermost layers of the nerve-fibres, as they pass over into the retina, become infiltrated, and the demarkations made by the choroidal opening and the scleral ring are covered up, so that the position of the disk is often only marked by the convergence of the vessels at the porus. There is, however, as a rule, no change in the level of the disk, nor is there much development of hypertrophied or altered tissue. On the contrary, the grayish infiltration extends pretty evenly over the surface of the disk until it reaches and merges into that of the retina. There is a well-defined striping, or radiation, of the tissue immediately surrounding the head of the nerve, when the retinal congestion before spoken of is present in the inner layers, but this is sometimes wanting when the œdematous character of the trouble seems to predominate over the congestive. Under these conditions the nerve and its immediate neighborhood seem to be absolutely devoid of small vessels, and the disk then seems to have a soft or soaked appearance, which is very different from what it offers in hyperæmia or irritation of the retina.

Hæmorrhages in this form of retinitis are not so common as in some other varieties, and when they do occur they are usually of small extent and of the striated form, corresponding to the direction of the nerve-fibres and following the course of the vessels.

Changes at the macula lutea are also comparatively rare, and when they do occur they are usually small in degree and correspond to the region of the yellow spot itself. The yellowish-white plaques, so common in renal retinitis, and especially the star-shaped figuration at the macula, are seldom seen in diffuse retinitis, especially in that form which is confined to the inner layers. When, therefore, these yellowish plaques are present, it usually shows that the trouble is not in the inner, but in the outer layers, or that both the inner and outer are affected.

There is, as a rule, but little general swelling of the tissue, although the effects of the inflammation may extend up to the anterior limits of the retina.

The neighboring portions of the vitreous may be involved in such a way that all the vessels of the fundus are more or less concealed from view. This is especially the case where the retinitis is

due to syphilis, and then the trouble often extends farther into the vitreous humor, in the shape of finely denuded and diffuse opacities.

These ophthalmoscopic appearances may last for a long time, or be of a temporary character and fade rapidly away, to return again in the same manner, after a longer or shorter interval. On the other hand, the trouble may end in atrophy of the retina, with or without repeated hæmorrhages into the vitreous, with formation of membranes, which sometimes become vascularized, and which may project a long way into the vitreous body.

Nothing can be predicated as to the amount of vision from the ophthalmoscopic appearances, as they may be most threatening, and yet the deterioration be slight, or they may be hardly perceptible and yet vision very much reduced; but when it is so, the outer layers of the retina are usually affected.

Diffuse Retinitis of the Outer Layers of the Retina.—This differs principally from that of the inner layers in the fact that the disturbances in the retina are posterior to the plane of the larger vessels, and that the veins are not concealed from view, and can be as plainly followed, as a rule, as the arteries. Moreover, the infiltration or exudation is not of so grayish a tinge, but more yellowish in appearance, and thus the hæmorrhages, when they occur, are less drawn out and rounder in shape, and do not so closely follow the course of the vessels. When the pigment epithelium is implicated, which is not uncommon, there are also evidences, especially in the retrogressive stages of the presence of pigment, which is sometimes collected into small concrete masses, and at others gives the effect of a granular or shagreen surface, or a smutchy effect, as if the color had run. At times, without doubt, the trouble does really extend to the choroid proper, and then we have a form of disease which may be and is very properly called chorio-retinitis; but I am convinced that in many of these cases to which the name has been applied, the real seat of the disease is limited to the outer layers of the retina and the epithelial layer, which is no doubt capable of undergoiug an inflammatory process, with hypertrophy of the cells, which are transported into the various layers of the retina from without inward, so that, although we may have ophthalmoscopic appearances, which clearly show the characteristics of choroiditis disseminata, or retino-choroiditis, yet the initial disease would seem to be often, if not commonly, a diffuse retinitis, especially when this attacks the outer layers.

Microscopic Characteristics of Diffuse Retinitis.—So far as the microscope is concerned, diffuse retinitis attacks preferably the nerve-

fibre layer and ganglionic layer. The retina, and especially the nerve-fibre layer, becomes thickened, and the latter is infiltrated with lymph-corpuscles. There is also hypertrophy of the connective-tissue elements, and of the radial fibres, which may even pierce the limitans interna and form loops over the inner surface of the retina, from which probably the membranes which extend into the vitreous, and which in some instances become vascularized, are derived. Pigment-cells are sometimes found deposited in the hypertrophied fibres. As a result of the inflammation, especially of the chronic forms, the nerve-fibre layer and the ganglionic layers become atrophied. While these changes are going on, the outer layers suffer but little, as a rule. By long-continued hyperplasia of the connective tissue, however, they are apt to pass into a granular mass, and then be absorbed; so that, finally, only the connective-tissue staging remains. The layer of rods and cones is commonly but little altered, unless there is a separation of the retina, when it can either be much affected, or even entirely destroyed.

A layer of albuminous fluid is not uncommonly formed between the pigment layer and the rods and cones; or between these and the limitans externa. In that form of retinitis that attacks the outer layers first, the changes that take place are principally an interstitial hyperplasia of the connective tissue of the granular layers, destruction of the rods and cones and pigment layer, with a partial hypertrophy of the latter, and transmigration of the pigment-cells into the retina. Besides this thin layer of fluid between the rods and cones, or between this and the limitans externa, there can also be a gluing together between the retina and pigment epithelium by means of a coagulable exudation that changes itself into connective tissue, and which by its contraction forms the atrophic spots so commonly seen in chorio-retinitis (Leber).

Circumscribed Retinitis.—This is a convenient name for that form of retinitis in which the symptoms, like those in the diffuse variety, are rather of a mild type, but where the grayish or yellowish-white infiltration is limited to a particular part or parts of the retina. The infiltration is inclined to extend in a longitudinal manner along the principal vessels, if in the inner layers; if in the outer, it is of a more circular form and of a more yellowish tinge. There is, as a rule, very little increased redness of the disk, and but few if any signs, either upon the surface or the neighborhood of the papilla, unless this has been the seat of a protracted and chronic form of inflammation; and then sometimes the larger vessels are bordered to a slight degree with white lines, or their walls are more accentuated in

color so as to become visible to the mirror. If the disease is of long standing, all the layers of the retina gradually participate in the trouble, and the grayish infiltration by degrees assumes a white hue along the borders of which there may be collections of pigment, and which may go on until there is complete atrophy of the choroidal tissue, allowing the sclera to shine through it. The disease then may be fairly called retino-choroiditis. This look of complete atrophy may take place without a corresponding decrease in the vision, which may be unexpectedly good, even over the affected portion, so that the scotoma, which is clearly present upon a careful examination, gives but little or no inconvenience to the patient, especially if the atrophic spot is in the periphery of the field. When,

FIG. 35.

however, the trouble is situated, as it not infrequently is, at the macula lutea, the central vision is very much affected, and the scotoma well defined, sometimes to a degree of total loss of vision in the region of the defect.

There is a form of circumscribed retinitis which is of great interest and importance, as it at times simulates the retinitis of Bright's disease so closely as to lead to the belief that there must be renal trouble. Repeated examinations of the urine, however, made even at long intervals between each other, show that this is and remains normal; and the only alternatives are, either that there are cases of retinis of a renal character, which show themselves in the fundus of the eye before the disease can be detected through the urine, or

that there is, in some rare cases, a form of local retinitis which closely resembles that of Bright's disease.

This form of retinitis shows itself in the shape of small, yellowish-white flecks or spots of a roundish shape, which are grouped together in clusters. These are evidently in the outer layers of the retina, and are usually situated in the neighborhood of the macula, or between it and the nerve. When at the macula, they are sometimes arranged in the star-shaped form so commonly met with in Bright's disease, and there are also the same changes in the pigmentation of the region. When situated near the nerve, the smaller spots sometimes coalesce and form round the nerve upon the temporal side a well-shaped crescent, which is so like that of the ordinary conus as to lead to the belief, at least upon a cursory examination, that there is a high degree of posterior staphyloma when the eye is really emmetropic or hypermetropic to a high degree. With a little care, however, it is seen at once that the color is entirely different, being of a dull straw-color; and that the choroidal tissue, instead of being atrophied, is simply covered up by an exudation or tissue-change, that lies in front of it. A remarkable point in the disease is the want of any active inflammatory condition, and especially the absence of any vascularity, swelling, or œdema; vision may or may not be much affected. In one or two cases, in advanced age, I have seen it nearly obliterated.

This form of retinitis occurs usually in elderly people, and is probably due to some diseased condition of the walls of the vessels. But I have seen it sometimes in young people, notably in the case of a young unmarried woman, in which repeated examinations failed to detect any kidney trouble whatever. Still, on the other hand, I have seen a case in which these changes were observed in the retina for three years, and where, during all this time, there were no abnormal symptoms in the urine, but where finally Bright's disease did develop itself. It thus becomes a matter of great interest, in respect to the fact whether disease of the kidney does not sometimes show itself in the eye months, or even years, before its presence is suspected in the urine.

Circumscribed Retinitis of the Outer Layers. — This is a peculiar form of circumscribed retinitis which consists of an increased thickness of the outer layers, and which is restricted to certain limited portions of the retina. The increased thickness is produced by a hypertrophy of the outer granular layers, with œdematous swelling, and changes at times in the layers of rods and cones, and pigment layer. This form of circumscribed retinitis occurs most fre-

quently immediately around the optic nerve, where it occasions a ridge-like swelling, like a wall surrounding the outer border of the disk.

This form occurs especially in syphilitic retinitis, and also, as will be seen later, in choked disk, where the outer layers are thickened, and split-like openings occur in the retina, which are filled with œdematous fluid.

Another rare form of this kind of retinitis occurs in the region of the yellow spot, which then becomes so untransparent as to reveal itself to the mirror. There is little or no change in the natural color of the retina, however, and the affected portion has not the soft look which accompanies ordinary retinitis. The mound-like swelling, which is an anatomical element of the part (see Vol. I, p. 32, *et al.*), appears to be increased in thickness, which may amount, as measured by the ophthalmoscope, to one or more dioptrics. The inner surface of the tumefaction thus formed, is always smooth and even, as if it had been simply pushed forward by the increasing substance of the outer layers. The outer surface must, however, at times be elevated into ridges or furrows by the uneven projection outwards of the swollen tissue. The epithelial layer then follows the inequalities, and is displaced from its natural position, which is shown by the play of shadows, as the eye of the patient undergoes slight movements. The black lines then formed in one place in the furrows, and uniting in another, change from a black hue to a dull gray and back again to black, when the eye returns to its original position.

The vessels of the part appear to be increased in size, while either vessels of new formation are present or those which previously existed have become so enlarged as to become visible. A marked feature in some of these cases is the peculiar appearance which the central portion of the region of the macula and fovea present. This latter seems to be so sharply defined that it offers the appearance of a clean-cut opening—as if, indeed, the tissue had been cut at this place with a punch. There is, however, no want of continuity of the retinal surface, and the effect is simply due to the exaggeration, through swelling, of the natural curves which enter into the formation of this part of the retina. I have seen cases of this description which had indeed been diagnosticated as rupture of the retina at the fovea. The tumefaction may, moreover, lead to the suspicion that a morbid growth is forming, which is only removed by the cessation of the process and the entire subsidence of the swelling. There is, as a rule, the usual central scotoma, with metamorphopsia and micropsia.

Not infrequently in myopic, and occasionally in other eyes, there

occurs a peculiar form of retinitis right at the macula lutea, that would seem not only to be in the outer layers of the retina, but also in the pigment layer itself—as if, indeed, exactly at this spot there had been a development of pigment—a sort of hypertrophy of the pigment-cell—a pigmentitis, if such a word can be used. This then shows itself, by a collection of pigment at and around the fovea, accompanied by little or no other inflammatory symptoms (see Fig. 36, also chromo-lithograph, Plate V, Fig. I). The pigment is usu-

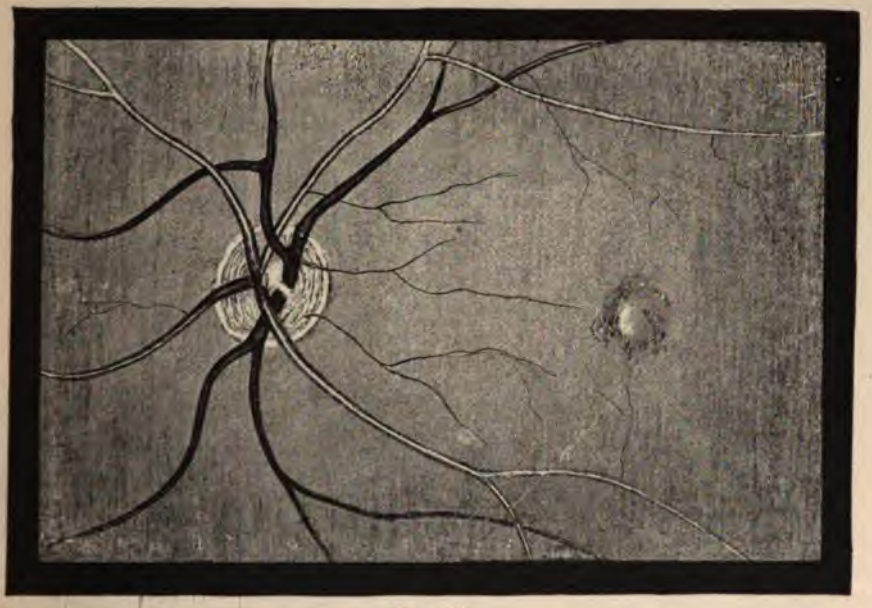

FIG. 36.

ally collected in a circular mass of a greater or lesser density, or it may present a reticulated shape similar to that of the coarser forms of black lace.

It has usually been assumed that this pigment was the result of a preceding hæmorrhage, from the fact that with inflammations at the macula we sometimes find, especially in myopic eyes, hæmorrhage associated with the pigment changes (Vol. I, frontispiece). There is, however, but little or no doubt that the pigment changes can take place without any occurrence of hæmorrhage at all. That in these cases the pigment is due to retinal disease, and is not of choroidal origin, is shown by the disturbance of the central vision and the peculiar distortion of straight lines. It is shown, also, by the ophthalmoscopic appearances, and by those of the micro-

scope, which shows changes in the outer layers of the retina and pigment layer.

The inflammation may, as it often does, extend to the choroid, and then we have the appearances of a spot, chorio-retinal disease with choroidal exudation or atrophy and the lustrous surface of the sclera.

Care should be taken not to confound the disturbances of pigment due to some light form of choroidal disease, or, indeed, to some congenital peculiarity, with the retinal affection mentioned above. This can usually be told from the disturbed state of the vision.

Rarer even than this form of pigmented change at the macula is a small—sometimes, indeed, very small—circular spot of exudation at the fovea and macula. This then, like other spots of exudation, has a grayish or yellowish color, and produces a defined and limited scotoma. These spots usually leave a lasting defect in vision, but they may occasionally be re-absorbed with very little or no impairment of sight.

Sometimes instead of a single spot there may be one small central spot just at the fovea, surrounded by a circle of still more minute

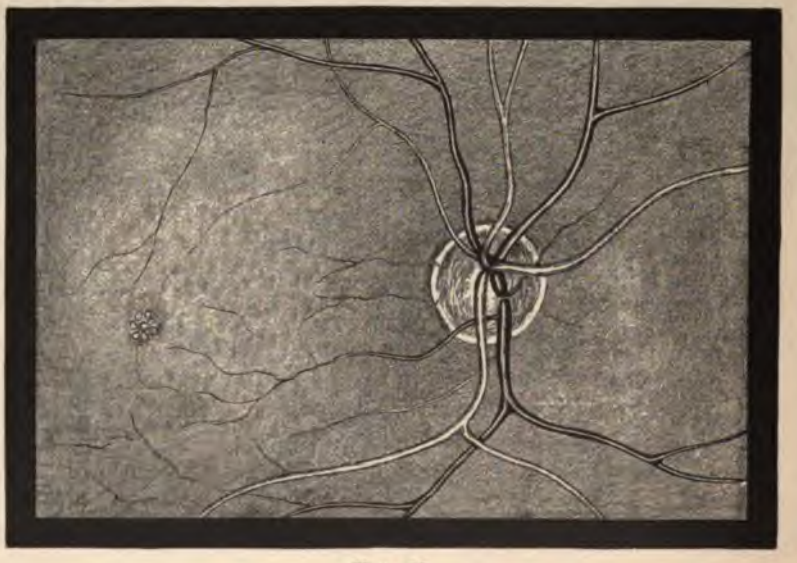

FIG. 37.

spots of a light yellow color (see Fig. 37, also chromo-lithograph, Plate V, Fig. II). [See also those shown in Jaeger's "Hand Atlas,"

Taf. xx, Fig. 89.] These spots sometimes give forth a glittering reflex, as if they were due to some lustrous substance. These cases should not be confounded with the somewhat similar pin-point spots which occasionally occur in the region of the yellow spot in eyes that are perfectly sound, or at least in eyes in which there is no disturbance of vision or other abnormity.

Relapsing Central Retinitis.—This peculiar form of retinitis was first pointed out by Von Graefe, and usually occurs in syphilis, but may have a non-specific origin. It is found in the latter stages of syphilitic disease, and is marked by a sudden obscuration of central vision, which may last only a few days or continue for months, when an improvement follows, or even apparently at first a cure, only to relapse again and again. In the first stages of the disease the patient suddenly complains of a loss of central vision, which is shown to consist of an irregular scotoma.

The mirror reveals a light opacity at the macula of a grayish or yellowish color, with groups of small and delicate punctate masses. There is but little disturbance of the disk. In the first attacks the ophthalmoscopic appearances usually pass entirely away. Little by little, however, changes occur at the macula, which become permanent, and show themselves by disturbances in the pigment and changes in the stroma of the choroid.

Retinitis Punctata Albescens.—This is a form of circumscribed retinitis which in later years has acquired a distinctive appellation of its own, although the writer has occasionally seen cases of it for the past twenty years, and looked upon it as a certain form of circumscribed or punctate retinitis. Since Mooren, however, gave it a name, several cases have been reported in literature under the above title, and not a few shown at the various clinics and societies.

The principal features of the disease consist in the presence of a great number of very minute dots of a whitish color (see Fig. 38, also chromo-lithograph, Plate VI., Fig. 1), so minute, indeed, that in some cases they can only be seen with the upright image; and then are so small and so closely packed together as to give the appearance of a stippled surface of a yellowish-white color. At other times the spots are larger and at greater intervals from each other. The spots themselves are usually round, but may be at times of an oval form. They are almost always situated between the disk and the macula lutea, although I have seen a case where the entire fundus seemed studded with minute white dots.

There are usually very few signs of active inflammation, although at times there is some vascular disturbance, some increased tortuosity,

especially of the veins, and at others some small hæmorrhages; or, in other words, there may be associated with the spots the symptoms of

FIG. 38.

a mild form of retinitis. Vision is commonly more or less affected, and there is usually a central scotoma when the macula is involved. Dr. Burnett has related a case, and given a drawing of the ophthalmoscopic appearances; mentioning, also, the cases reported by Mooren, Kuhnt, and Hirschberg ("Archives of Ophthalmology," vol. xii, Vol. I, p. 22).

Retinitis Hæmorrhagica, or Apoplectia.—Strictly speaking, this is not a form of retinitis, but rather a symptom than a disease, since hæmorrhage is common to all forms of retinal inflammation. Moreover, it is usually due not to a disturbance of the retinal tissue, but is a disease of the walls of the vessels themselves, which favors the transudation of the blood-corpuscles. It is rather, then, a vasculitis than a retinitis. When, therefore, signs of retinitis are present, these are usually secondary and due to the irritation caused by the blood-clots, which may lead to a diffuse disturbance in the retina, to foci of fatty degeneration, and to varicose hypertrophy of the nerve-fibres; also, occasionally, to acute glaucomatous attacks. Care should be taken also to draw a distinction between simple hæmorrhagic extravasation

into the retina and phlebitis with thrombosis, which may be accompanied by all the signs of the most active inflammation, as shown elsewhere.

A striking peculiarity of so-called retinitis hæmorrhagica is the great diminution in the diameters of the arteries; so much so, that the wonder arises where the supply for such profuse and repeated hæmorrhages could possibly come from (see Fig. 39, which is from Liebreich).

Upon looking upon a well-marked case of retinitis hæmorrhagica, the conviction is almost irresistible that the bleeding is due to rupture of the walls of the vessels; but here, as in other hæmorrhages into the retina, the microscope does not show any break in the walls, and the bleeding is consequently to be looked upon as the result of diapedesis rather than rhexis; although this may, and no doubt does, occasionally occur, at least as the result of traumatic rupture of the globe.

FIG. 39.

As a rule, the hæmorrhages are from the larger vessels, and are situated near the nerve, and may vary in a marked degree in number, size, and shape. In simple retinitis hæmorrhagica, the extravasation begins to grow pale at the edges of the clot and not in the centre, as is the case in septic retinitis and other forms of retinitis, where the composition of the blood is altered as to the proportion of its component parts. (See Liebreich's "Atlas D'Ophthalmoscopie," Tab. viii, Fig. 1, *et seq.*)

When, as sometimes occurs, white spots are formed in the retinal tissue by fatty degeneration or through varicose hypertrophy of the nerve-fibres, there is considerable similarity between this form of retinitis hæmorrhagica and retinitis albuminurica.

The disease usually occurs in elderly people, and is frequently met with in connection with diseases of the circulation and valvular troubles of the heart, hypertrophy of the left ventricle, and aneurisms.

Retinitis hæmorrhagica may occur in both eyes; when it occurs in one eye, it is usually associated with disease of the heart. It is also often the forerunner of cerebral apoplexy, although the disease may last for years without any signs of heart-disease, and without being accompanied by any cerebral or other constitutional disturbance.

Syphilitic Retinitis.—The infection due to syphilis may be accompanied or followed by any form of retinitis. It may attack the outer layers or the inner; may be circumscribed or diffuse, though it is usually the latter. It may, moreover, be associated with neuritis, forming genuine neuro-retinitis, which, when accompanied by gummata of the brain, may express itself in the most violent manner, such as it is common to find with cerebral tumor. Syphilitic retinitis may be associated, and often is, with choroiditis, producing a retino-choroiditis of a typical form. Still, notwithstanding this comprehensive character of the disease, there are some peculiarities which occur so frequently with it as to be somewhat of an aid in differential diagnosis, and which lead to the employment of specific remedies that might not otherwise have been adopted.

In the first place, the retinitis is usually of a diffuse form, in which there is little if any change in vascularity. The disturbance is usually near the nerve, and follows the larger vessels, shooting along these at times in a comet-like way. This form of retinitis is almost always accompanied by troubles in the vitreous of a diffuse nature, and which consist of dust-like aggregations, with now and then a thin, sparse membrane. Hæmorrhages are comparatively rare, although there are cases in which they not only occur, but are profuse. On the other

hand, iritis, which is usually rare in other forms of retinitis, is not uncommon in the specific varieties. Irido-cyclitis is also very apt to occur with either serous or plastic exudations. Some authors mention the fact that, besides the more gentle elevations of the surface of the retina, that occur when the external layers are increased in thickness, and which may happen in ordinary retinitis, there may be in this variety organic masses of considerable size which may project for quite a distance into the vitreous, and which later by secondary contraction turn into white cicatrices of a radiating form. These might possibly be of the nature of gummata. I have, however, never seen such growths.

Syphilitic retinitis often passes into chorio-retinitis, especially when it begins in the outer layers of the retina, and marked changes sometimes follow even when the retinal disturbance has seemed with the ophthalmoscope to be very slight. Thus, in the later stages, and as a retrogressive appearance, there often comes a diffuse paling of the pigment layer, with the development of flecks of pigment, which differ from those of retinitis pigmentosa (see sketch at bottom of Fig. 40), inasmuch as they do not have the characteristic bone-corpuscle shape; nor do they correspond so nearly with the course of the vessels and perivascular spaces, although occasionally not only does the trouble assume the appearances of a true retinitis pigmentosa, but does indeed turn into it as a secondary result.

Through the atrophy of the epithelial layer the fundus acquires all the appearances of a diffuse choroiditis, with marked changes in the pigment and stroma. (See Fig. 40, also chromo-lithograph Plate VI, Fig. II).

From the considerable changes which occur in the choroid, and the comparatively slight disturbance in the retina, it might be inferred that the trouble in these cases began in the choroid, and it has been classed by some authors as syphilitic choroiditis. There is no doubt but that this is sometimes the case, but the origin of the disease is nevertheless usually in the outer layers of the retina.

Besides this diffuse retinitis, which is so characteristic of syphilis, we sometimes get a true plastic retinitis, which then shows itself in circumscribed spots of a yellowish-gray color. These spots may extend for a considerable distance along the vessels, or they may be of a small, round form, and be either in circumscribed localities, especially in and around the macula, or strewed over the entire retina, and sometimes so closely aggregated together as to give to the retina a stippled appearance, resembling that sometimes seen in retinitis albescens.

FIG. 40.

The vision is sometimes very much reduced in specific retinitis. It often improves to a considerable degree only to fail again when secondary changes occur, and this is especially the case in central relapsing retinitis due to syphilis. As in other forms of retinitis, atrophy of the membrane to a greater or lesser degree may occur, with reduction in the size of the vessels and paling of the disk. This, however, is not always the case, and we are sometimes astonished to find so great a reduction of sight, after the attack has passed off, with so comparatively a normal fundus. When the trouble is in the outer layers of the retina, we have the usual torpor of the retina so common in all forms of retinitis when these layers are affected. With this torpor retinæ there are peculiarities of the field of vision which are expressed by the ordinary and ring scotomata, which are very different from the contractions of the field, when the stem of the nerve is affected. The color-sense is not usually affected, unless there is some atrophy of the optic nerve.

Renal Retinitis.—Nephritic retinitis, retinitis albuminurica, or the retinitis of Bright's disease, as it is variously called, may occur with any form of kidney-disease, although it occurs most frequently with the contracted kidney. It is found also with the large, white kidney, and with lardaceous degeneration of the kidney, but especially, if not solely, in the chronic forms of these diseases. The ocular changes are usually associated sooner or later with hypertrophy of the heart, and it is only in exceptional cases that it is not. They usually occur only where there is real organic disease of the kidney, although it has been asserted, by Eales and others, that they may be found in young people in that variety of cases which are to be classed as temporary and functional disturbances, in which for the time being albumen is present in the urine.

In the earlier stages of the disease the changes may present themselves in a variety of forms which have nothing distinctive in them. They may take the form of an ordinary circumscribed retinitis of a mild form, or of a retinitis hæmorrhagica, since extravasation forms a conspicuous part of all renal retinitis. I have seen cases where the only ophthalmoscopic sign was a single small spot of hæmorrhage or exudation, without a trace of vascular disturbance upon the disk or surface of the retina. In the early stages there is but little if any swelling of the tissue. In common with other forms of retinitis the arteries are somewhat reduced in size, although both they and the veins seldom show tortuosity, or twist to any considerable degree. The reduction in the size of the arteries is probably due to contraction which takes place in the smaller vessels in Bright's disease, and which leads to hypertrophy of the muscular coat, and to decrease in the lumen of the vessel by formation of a tissue, by which it may be entirely obliterated.

When retinitis albuminurica has assumed a distinctive and characteristic appearance it has usually passed from a state of simple retinitis to that of neuro-retinitis, and it is really under that form that it is oftenest seen with the mirror. When thus seen its chief characteristics are shown by the presence of large yellow masses (see Fig. 41, also chromo-lithograph, Plate IV, Fig. II), which are commonly situated near the optic disk, and arranged more or less in a circular form round the papilla, but at a little distance from it. This zonular character of the white plaques differs from that of other forms of retinitis in which the exudation and degeneration are less diffuse and more longitudinal in extent, and which correspond more closely with the direction of the larger vessels. Near the disk these white masses have a tendency to coalesce, and the

papilla then becomes the centre of what appears to be an enormous plaque of exudation.

It is, however, at the macula that the most characteristic signs occur. These consist in the presence of a collection of circumscribed

FIG. 41.

minute dots surrounding the macula, or by a star-shaped figure of a radiating character. These signs are almost pathognomonic of Bright's disease, though it must be admitted that they are sometimes met with in the secondary or retrogressive stages of other forms of retinitis and neuro-retinitis. It is the constancy of its occurrence and its presence in the earlier or progressive stages of the trouble which are characteristic of Bright's disease, besides the fact that the color of the masses is softer and more yellowish than when they are the result of secondary changes, for at this time they have a whiter and more cicatricial aspect.

It is not yet known exactly to what these dots and star-shaped figures owe their peculiar appearance, but it is probably due to fatty degeneration of the tissue, and principally, according to Schweigger and others, to that of the radial fibres which end here. It would seem, also, as if fatty degeneration and sclerosis of the capillaries and

small vessels in which this region abounds, with hypertrophy of the connective-tissue elements, must contribute largely, if not principally, toward the production of the appearances. (See Vol. I, pp. 35, 36.)

The stellate figure sometimes seems to rest upon a dark background, or the yellow radiations to alternate with those of a dark-brown color. This, according to most observers, shows an alteration in the pigment layer. From the peculiar radiating direction of these dark stripes, however, it might also be a question whether they were not due to alteration of the hæmatin in the blood of the diseased smaller vessels and capillaries; especially as, after long lapses of time, the brown color often disappears entirely, leaving only small white dots and minute white striæ.

The large, white plaques, so characteristic of this form of retinitis, consist of regions of hypertrophied nerve-fibres and fatty degeneration, and it is sometimes a little difficult to make a distinction between the two. The hypertrophied fibres, however, lie in the innermost layers, while the fatty degeneration is principally in the middle and outer granular layers. This can often readily be told by the fact that the vessels run unimpeded and unconcealed over the white plaques, showing that they are in a posterior plane. This is clearly shown in Liebreich's "Atlas," Tab. ix, Fig. 1. There is a difference, too, in color and texture. The hypertrophied nerve-fibres, especially in the later stages, have a grayish tinge and a firmer look, while the fatty degeneration is more yellow and of a softer and less condensed appearance; and where the two exist together, as is not infrequently the case in the inner layers, the contrast is more marked.

Hæmorrhages may take place in any stage of Bright's disease. They are commonly situated in the inner layers, and have consequently oftentimes a radiating appearance. They may, however, especially in the later stages, have a circular form, and then have a white centre, with a red circle surrounding it. Small punctate extravasations occur sometimes as an early symptom in the deeper layers of the retina, and when this takes place the prognosis is apt to be less favorable than when the bleeding is from the larger vessels.

Retinitis albuminurica may suddenly become, or gradually pass into, a violent form of neuro-retinitis, which may simulate that due to cerebral causes so exactly as to offer but few if any distinctions from it. Still, as a rule there are some distinguishing marks which serve to point out the true nature of the disease. Thus, in the earlier stages of retinitis from Bright's disease, the hyperæmia is more apparent in the smaller vessels of the disk, which produces a more diffuse redness of the papilla, the larger vessels being very little, and often not at all,

affected. In choked disk, on the contrary, from cerebral disease, the larger vessels are the first to show any signs of congestion, especially the veins, which are often increased in their diameter and have an increased tortuosity, which is sometimes extremely marked even in the very beginning of the trouble. Tissue changes also occur in the inner layers of the retina much earlier in Bright's disease, than in choked disk dependent upon cerebral disturbances. The white plaques are also more numerous, more apt to coalesce, and hence have a larger superficial extent in retinitis albuminurica, than in choked disk. The swelling of the nerve and the surrounding tissue is less in Bright's disease, and fades more evenly off toward the periphery, while in choked disk the head of the nerve protrudes to a greater degree and joins the retina with a rather sharp declivity, forming a sort of circular plateau, over the edges of which the vessels dip down into the general level of the retina. This mushroom form of the papilla is quite characteristic of tumor of the brain, and when combined with an excessive elevation, as, for example, from 7 to 9 D., is nearly always a sure indication of a growth of some kind in the brain. The matter, however, is of less importance as a simple examination of the urine is usually sufficient to establish a diagnosis. Still, it is not always so, as the changes in the eye may in some cases of granular kidney precede, even for a long time, the presence of albumen in the urine, and it is therefore well to bear in mind the preceding distinctions, as they hold good in a differential point of view between tumor of the brain and other forms of retinitis.

Retrogressive Appearances.—In the retrogressive stage of retinitis albuminurica, like that of other forms of retinitis, the changes can disappear completely, or they can come to a standstill, or relapses may occur even after an improvement has taken place. On the other hand, the ophthalmoscopic appearances may become less and less, while at the same time the disease is progressing. Of the plaques, those at the macula lutea are the last to disappear, and these can remain after all other changes have faded away, or, according to some authorities, may never disappear. White, glistering spots, probably due to cholesterine, sometimes occur in the plaques, while at others glittering points are seen that have the appearance of minute deposits of lime.

As the secondary changes progress, the papilla gets more and more discolored, becomes again sharply defined, and may gradually pass into a condition of atrophy more or less complete, while the vessels show the usual reduction in diameter, with signs of hyperplasia of the adentitia. Sometimes in this stage of the trouble there are marked

changes in the epithelial layer, and the fundus assumes the appearances seen in diffuse chorio-retinitis, showing that the outer layers of the retina and the epithelial layer, if not the choroid itself, have participated in the disease.

Nephritic Retinitis of Pregnancy.—This does not differ from that due to ordinary Bright's disease, as they are both due to the same cause, since the retinitis of pregnancy is not caused simply by stasis hyperæmia, but is a true nephritis. Neither is the retinitis due to the presence of the albumen alone, as there are cases of true nephritic retinitis, in which there is not a trace of albumen or casts of any description in the urine; while, on the other hand, there are cases where albumen exists, and where there is no retinitis. Here, as elsewhere, the inflammation in the retina would appear to be due to an altered condition of the blood which produced a disease of the walls of the vessels. Its true nature, however, is not yet determined.

As a complication of the retinitis we sometimes have an implication of the choroid, which shows itself by the changes above alluded to, of diffuse chorio-retinitis; also, though more rarely, by those of choroiditis disseminata, and more rarely still of choroidal hæmorrhage. Hæmorrhage into the vitreus, it is true, is not so very rare, but when it occurs it is usually, contrary to the general opinion, from the retina and not from the choroid. There may be, too, embolism or thrombosis of the vessels—also separation of the retina.

In retinitis albuminurica, the vision may be more or less affected, ranging from hardly a perceptible deterioration to complete blindness, especially when combined with uræmic poisoning. There is usually a defect in the field, though sometimes central vision is defective from changes or hæmorrhage at the macula.

Uræmic Amaurosis.—Uræmic blindness is not infrequent in the nephritis which accompanies or follows scarlet fever, or serious cases of other exanthemata, and in pregnancy. The blindness is usually temporary, and not accompanied with opththalmoscopic signs, as the trouble is either of too transient a nature or the patient succumbs before retinal changes have had time to occur. In pregnancy, however, it sometimes happens that repeated attacks of uræmic blindness occur, and that they take place with some women in successive pregnancies. In these cases, after a lapse of a longer or shorter time, there are often unmistakable signs of white atrophy of the optic nerve, with limitation of the field of vision. Cases have been reported where the women have become totally blind through these attacks in successive pregnancies; and the writer has reported a case where the patient was saved from the loss of her remaining eye by

the production of premature delivery. The other eye had become totally blind in former pregnancies.*

Diabetic Retinitis.—Retinal affections are comparatively rare in diabetes, and belong, as a rule, to the graver forms of the disease. They occur far less frequently than troubles in the lens, and usually do not make their appearance until the disease has lasted a long time. The diabetic form of retinitis offers but little which is distinctive of presence of sugar in the urine. As a rule, however, there is more hæmorrhage and less exudation or white plaques than in retinitis albuminurica, and the spots are smaller, and not so apt to be situated at the macula, or grouped in a zonular manner round the papilla. Dr. Noyes,† however, many years ago reported a case, as others have since, in which the appearances were absolutely identical with those seen in chronic renal disease. In this case of Dr. Noyes's, repeated examinations of the urine, extending over considerable time, and made by different observers, failed to discover any albumen in the water. Dr. Culburtson published a case in which there were marked symptoms of choked disk, which may, in very rare cases, happen here as with retinitis albuminurica. Jaeger gives a drawing in his Atlas (Fig. 64, Tab. XIII), which represents a case of retinitis in a diabetic patient, and which, although it lacks some of what are considered the most characteristic marks of retinitis albuminurica, in the absence of the changes round the macula, and the zonular exudation round the nerve, yet which, nevertheless, are so characteristic of renal retinitis as to call the attention at once to the necessity of an examination either for albumen or sugar.

Here, too, as with Bright's retinitis, hæmorrhagic glaucoma may occur. Occasionally it has happened also that diabetes has been associated with tumor of the brain, and the optic neuritis has been attributed to the diabetes, until the autopsy revealed the true origin of the eye symptoms. This might well happen also in those cases of retinitis albuminurica in which the ophthalmoscopic appearances resemble choked disk so closely, and where no autopsy was made as to the condition of the cranium. Diabetic retinitis, like that of Bright's disease and kindred affections, usually occurs in both eyes. I have, however, seen a case where it existed in one eye only for nearly a year without the other being affected at all, as long as the patient was under observation.

There is a form of amblyopia that occurs in diabetes in which

* "Premature Delivery for the Prevention of Blindness." Trans. Amer. Ophth. Soc., 1882, p. 423.

† "Trans. Amer. Ophth. Soc.," 1868, p. 71.

there is a central scotoma. Here the visual symptoms bear a strong resemblance to that form of nerve-disease which has been classed as a post-ocular neuritis, while the ophthalmoscopic appearances have a great similarity to those seen in amblyopia potatorum and tobacco amaurosis, both of which, though classed as a disease of the nerve, nevertheless show some delicate changes in the retina itself. These changes consist in this form of amblyopia from diabetes, of a delicate, hazy appearance of the whole fundus which at times is very striking. In the early stages of the disease the nerve has a delicate but perceptibly increased vascularity which expresses itself in a mild form of hyperæmia which later on disappears and gives place to the signs of beginning atrophy. The light-streak becomes diminished, or is entirely wanting upon the larger vessels, and later there are the signs of a mild form of perivasculitis. I am of the opinion, therefore, that many of the cases which have been classed as primary atrophy in diabetes are really the secondary results of a low form of neuro-retinitis, and that the vessels of the retina are affected as well as the tissue of the nerve. In these cases, as with all nerve disease, the color-sense is affected, which is not the case in the simple forms of retinitis.

Retinitis of a hæmorrhagic type, like that of Bright's disease, may occur in diabetes insipidus.

Oxaluria.—This condition is sometimes accompanied by changes in the retina and vitreous which would point to alterations in the blood-vessels and subsequent hæmorrhages, which are sometimes so numerous and extensive as to suggest retinitis hæmorrhagica with small spots of fatty degeneration. There is also, at times, a hyperplasia of connective tissue which is probably due to multiple hæmorrhages with a constant tendency to relapse, and which so often produces such hyperplasia in other forms of retinitis. Jaeger reports cases where, after the clearing up of the vitreous there was diffuse retinitis, with the development of fine vascular loops in the vitreous.

Retinitis may also be excited by exposure to the fumes of osmic acid.

Splenic Retinitis.—Leukæmic retinitis may resemble the ordinary forms of retinitis of the diffuse type, but when developed it presents a very characteristic picture, the principal features of which are the peculiar color of the background of the eye and that of the blood in the retinal vessels. The color of the fundus is of a decided though usually pale orange tinge. This shows that the blood in the choroidal vessels varies from its normal tint, and points to the conclusion that it must vary also in all the other vessels of the body, although often-

times the complexion of the face does not show it. This orange tint not only remains under an examination by daylight, but, according to Becker, is more conspicuous with it than with lamp-light. Liebreich, who first discovered the disease, gives a striking representation of this peculiar orange shade.*

This orange tinge is, however, by no means constant, and did not exist at all in the majority of cases seen by the writer. On the contrary, the color most frequently observed was a pale grayish-pink, and in some cases no deviation from the normal fundus in regard to color or shade was noticed.

The color in the retinal veins is usually of a bluish-pink, while that in the arteries is of the same orange color noticed in the fundus, although lighter in shade.

A marked peculiarity is the predominating tortuosity of the vessels and the increase of their diameter. The veins are sometimes enormously distended, and double and return upon themselves to a degree which I have never seen in any other eye disease, resembling at times some of those rare and exaggerated forms of dilatation and tortuosity met with in cyanosis bulbi. A curious fact in connection with these dilated vessels is the absence of swelling of the tissue, as is shown from the want of protrusion and from the fact that the general curvature of the vessel remains in the plane of the retina, though, of course, in some cases, as with all retinitis, there is some antero-posterior bending of the vessel; but it is very little in comparison with the marked lateral and serpentine deviations.

In this as in other forms of retinitis the arteries are not nearly so much increased in diameter, and they may be even narrower than normal.

There is usually but little or no swelling of the disk, though in very rare and very marked cases there may be considerable protrusion of the head of the nerve. In a case described by Oeller this was due to œdema and a large collection of leucocytes in the papilla anterior to the lamina cribrosa.†

There is, moreover, little or no injection of the disk. On the contrary, sometimes the papilla seems paler than usual, as it often does in mild forms of retinitis of any description, probably because the capillaries are concealed by the œdema. The edges of the nerve are sometimes obscured, but the opacity is usually of a small amount. When present it is radiating in form and extends along the larger vessels, which at times are concealed by it. Often when there is no

* "Atlas d'Ophthalmoscopie," Tab. X., Fig. 3.

† "Archiv für Ophth.," XXIV., Band III., p. 245.

real opacity a lighter and band-like portion of the retina seems to run along the vessels, which is especially apt to come out under rotations of the mirror. This gives the impression that it was due to the presence of lymph-corpuscles, which the microscope shows are often collected in the lymph spaces and adventitia surrounding the vessels. Indeed, the whole fundus gives the idea of a sort of infiltration due to the accumulation of white corpuscles either in the vessels or tissue; and in fact in the case described by Becker the blood of the patient appeared when examined of a pale red color, and resembled bloody matter rather than human blood.*

Another peculiarity of splenic retinitis is the tendency to hæmorrhage, and the shape and position of the extravasations. According to Gowers, the tendency toward bleeding is greater in leucocythæmia than in anæmia, for it occurs in the former where the percentage of red corpuscles is greater than in anæmia, and in two cases he had met with retinal extravasations where the blood contained fifty per cent of red globules.† These hæmorrhages are usually of a circular form, and are situated either near the equator, or anterior to it; sometimes, however, near the macula, and between it and the nerve. They may be large or small, and, if of any considerable size, they often have a white centre surrounded by a ring of hæmorrhage or fine ecchymoses. The microscope shows that these spots are elevated above the surface of the retina, and consist in part of varicose hypertrophy of the nerve-fibres and of fatty degeneration, and in part of a collection of lymph-corpuscles. These hæmorrhages may disappear rapidly, or be supplemented by others in different parts of the fundus.

Sometimes splenic retinitis has many if not most of the characteristics of simple retinitis hæmorrhagica. Hæmorrhage may also occur into the vitreous.

Changes in the choroid may occur, and its tissue be found infiltrated with leucocytes, by which the tissue may be considerably swollen. This would, when it took place, account for the peculiar color of the background of the eye, and its absence in other cases where this infiltration had not taken place.

Exophthalmos has been observed as a rare incident, and due, it is supposed, to lymphoid growths in the orbit. It would seem more probable, in view of the tendency to hæmorrhage, that it might be due to a somewhat profuse bleeding.

Sclerosis of the Retina (*Pigmented and Non-pigmented Retinitis*). —Retinitis pigmentosa is a curious affection which attacks the retina,

* "Archives of Ophth.," vol. i., No. 1, p. 347.

† "Medical Ophthalmoscopy," second edition, p. 214.

and which is one of the most interesting of eye diseases, pathologically as well as ophthalmoscopically. It is characterized by the hypertrophy of the interstitial connective tissue of all the layers of the retina, with atrophy of the nervous elements and the migration of pigment, caused by a hypertrophy of the coloring matter of the epithelial layer, and which is collected principally along the retinal vessels. These changes in the coloring matter consist partly in a new formation of pigment-cells, which are very rich in pigment, and partly in the atrophy of the pigment, so that the coloring matter is very much reduced in quantity and shade, or may be even entirely wanting.

The changes which mark the disease usually begin in the outer layers of the retina, the nervous elements of which are crowded out by the increase in the connective tissue of the staging work until they finally disappear. Later the inter-granular layer also vanishes, and the outer coalesces with the inner granular layer. The rods and cones are destroyed, and at last the entire retina is changed into a nearly homogeneous network, in which the division into layers is almost wholly lost. At times the retina becomes adherent to the choroid in spots.

The changes in the pigment are also very striking. At the places where the choroid and retina have grown together there is a rich hypertrophy of pigment. The cells are here of a brownish-black color, and are thickly filled with large pigment granules, which are very irregular in shape and which lie in several super-imposed layers. Strings of this pigment stretch themselves into and through the various layers of the retina, which can be traced up to the nerve-fibre layer. These run at first perpendicular to the plane of the pigment layer, but finally take a parallel course along the larger retinal vessels.

The vessels show a remarkable thickening of their walls and a corresponding decrease in their lumen, by which, under the microscope they acquire a rather homogeneous and glittering yellow appearance in which the structural elements are not clearly defined.

Colloid thickenings take place ordinarily on the vitreous membrane of the choroid. These may extend far into the retina, even up to the fibre layer, and then their tips are covered over with pigment. The changes in the choroid are slight, though a certain degree of sclerosis of the vessels is not uncommon. This gives a yellowish tinge to the blood-column, probably from a thickening and alteration of the wall. Sometimes, too, the pigment is increased in the intervascular spaces. When this takes place there is a certain amount of irregularity in its disposition, which means that it is not

due to a physiological arrangement such as is known as the "choroid tigré." That portion of the vitreous which lies immediately next to the retina is often adherent to it, and contains a number of cell elements. The stem of the nerve is often atrophic up to and beyond the chiasma (Leber).

Such marked anatomical changes as these are necessarily accompanied by a striking ophthalmoscopic picture when the disease is seen at its height. At the beginning, however, the visible changes may be of a very moderate character. The first change noticed by an attentive observer consists in the narrowing of the vessels, or rather blood-column, which is then smaller in proportion nearer the nerve than toward the periphery, so that the vessels seem to carry a uniform diameter farther toward the periphery than in the normal eye, and look rather like small cylindric tubes than tapering vessels. As the disease progresses, the decrease in diameter becomes very marked until the vessels are reduced to thread-like dimensions. The transparency of the wall, though not much affected, must, however, suffer to a degree by the increase in thickness, as the light-streak is either much reduced in brilliancy, or even entirely wanting in vessels of a calibre where in the normal state it is perfectly visible. It is well to remind the reader here that the reduction in the size of the vessel, as seen by the mirror, is not due, at least in the first stages, to any decrease in the outside diameter of the vessel, but to a reduction in its lumen by the infiltration of a peculiar tissue which lines the inside of the vessel, and which gradually reduces the apparent diameter of the blood-column (see Vol. II, p. 25).

Sometimes, too, the wall of the vessel itself becomes more apparent, or it is lined by a fine white streak, such as is sometimes seen in moderate forms of perivasculitis, which shows that there is a low stage of vasculitis accompanying the sclerosis, although there is seldom or never any increased hyperæmia in the purer varieties. Not infrequently, however, when the retinitis pigmentosa is secondary to some other retinal or choroidal inflammation, there is a preceding hyperæmia, and sometimes an increased injection of the nerve, but this must be looked upon as an associated trouble, and not necessarily a part of the disease.

During the stage in which the larger vessels are becoming narrower the capillary vascularity of the disk begins to fade also, and the nerve assumes a whiter hue; not, however, the dead white of atrophy, but rather a creamy white; and, moreover, there is not the clean-cut tendinous aspect which characterizes simple atrophy, but rather an appearance of a waxy and untransparent substance which conceals the

lamina cribrosa from view. The edges of the nerve are only moderately defined, and they are sometimes bordered by masses of pigment, either arranged in flecks or in a more or less well-formed ring round it. It is very rare, however, that any pigment is found upon the surface of the papilla.

The most conspicuous ophthalmoscopic change, and one from which the name of retinitis pigmentosa is derived, is the collection of pigment in small masses in certain parts of the fundus. These aggregations usually begin in the anterior part of the field near the equator, and form a zone round this portion of the field, leaving a free space both toward the ora serrata and toward the macula lutea and disk. This even and zonular form of the pigmented region usually advances toward the posterior pole of the eye, as the disease progresses, with a uniform circumference, with few or no irregularities or defects in it. This method of progress is very characteristic of the disease. When, therefore, the observer finds that there are irregular defects in the field of vision, and especially if they assume a sector-like shape, he can be pretty sure that the trouble is not pure retinitis pigmentosa, but has either been preceded or accompanied by some choroidal or retino-choroidal affection. This is important in the way of diagnosis, for the more the trouble inclines toward a choroiditis the better is the prognosis.

It is only very rarely that pigment to any considerable amount is found directly in the macula lutea near the fovea, unless, indeed, the disease is associated with or the result of a specific retinitis or chorio-retinitis.

The form that the small collections of pigment take is very peculiar and very characteristic. It is always in comparatively small masses of a spindle- or star-shaped form, which show but little tendency to coalesce into larger ones, but which give the idea as if the stellate masses had just come together by their points, so as to form a delicate mesh-work.

These masses of pigment are sometimes so fine as to give the effect of a stippled surface, or a pepper-and-salt appearance. In the more fully developed cases the pigment assumes the well-known "bone-corpuscle" shape, which is almost pathognomonic of the trouble, and is never met with, so far as I know, to any degree in any other form of retinitis, although we do see occasionally in these an isolated configuration which resembles it pretty closely. The accumulation usually begins in the outer layers, which can be determined by the mirror as well as by the microscope. Later on it permeates all the layers and follows along the course of the vessels, and often lies in smaller

or larger masses on top of these. I have seen the wall completely surrounded by a delicate black open lace-work, which would sometimes extend for quite a distance. The pigment to form this was probably carried along the perivascular lymph spaces.

The pigment may even penetrate into the vitreous, and in the more marked cases there is often a small mass situated just behind the posterior pole of the lens, which careful examination shows is not really in the lens but posterior to it, and in the very spot which marks the centre of motion of the eye. (See Vol. I, p. 187.)

The pigment epithelial layer in the neighborhood of the diseased region of the retina, or even beyond it, is always affected; the borders of this part show a diminishing change until a normal-looking fundus is reached. At these places the epithelial layer has a macerated look, with specks of pigment strewed over the surface, while the intervascular spaces, from the lack of pigment in the epithelial layer, are somewhat more clearly marked. When the de-coloration is of considerable degree, there appears to be an alternating shade of yellow or yellowish-brown with the darker portion of the pigment, and occasionally small, round, and brilliant bodies are seen, which look like colloid formations, upon the vitreous membrane of the choroid. These spots do not, of course, correspond always with the pigmented portions of the retina, although they occasionally do, and when they are numerous and of a circular shape they bear a striking resemblance to chorio-retinitis or choroiditis areolaris.

In the purer forms of retinitis pigmentosa there is invariably a remarkable concentric narrowing of the field of vision, which progresses as the disease advances, until a minimum field is reached, which may not amount to more than a few degrees. This contraction of the field is almost invariably greater than what corresponds to the extent of the pigmented portion, showing that the percipient elements of the retina are affected before the infiltration of pigment takes place—indeed, in some cases the narrowing of the field occurs where there is no accumulation of pigment to be seen with the ophthalmoscope. On the other hand, there are still rarer cases where the mirror shows marked changes in the pigment layer and where the concentric narrowing of the field is wanting, or, still rarer even than these, there are cases where the central vision may be much affected without any concentric narrowing of the field.

There is almost always a great amount of torpor of the retina, which usually extends over the entire field, so that, in addition to the concentric limitation by daylight, there is usually night-blindness for the entire retina.

This pigment degeneration, which expresses itself as retinitis pigmentosa, may be either congenital or acquired. It is probably much oftener congenital than is supposed, but only shows itself later in life under some fortuitous circumstance, which lights up a progressive disease. When congenital it is well to remember that the pigment changes do not show themselves usually until the eighth or tenth year, and even later. In examining very young children I have often been struck with a condition of the fundus which, I imagine, is often the precursor of true retinitis pigmentosa, which may develop itself later in life. In these cases vision is usually defective, varying from about $\frac{1}{6}$ to $\frac{1}{4}$ of the normal amount. There is in these cases a suspicious air to the nerve, which looks opaque and untransparent beyond the years of the patient. The vessels are small, especially the arteries, in which the walls are sometimes more visible than should be the case at this time of life; or there may be just at the nerve fine white lines along the vessels. The most important changes even in these doubtful and incipient cases are those of the pigment, together with the general look of the fundus. This has a faded aspect, with a peculiar shagreen or pepper-and-salt appearance, due to a multitude of very fine pigment points scattered all over it. This sort of fundus is usually found in children who have all the evidences of congenital mal-nutrition, or who look like the prey to some hereditary taint. Great care should be taken that such eyes are not subjected to overstraining, particularly during early life; especially, too, as congenital pigment degeneration is not always progressive—not nearly so much so as the acquired.

Retinitis pigmentosa is almost always double-sided except when due to syphilis, which it is important to notice in a diagnostic point of view, as in the latter case the prognosis is more favorable.

Differential Diagnosis.—The only diseases with which retinitis pigmentosa would be at all likely to be confounded are those due to choroidal and chorio-retinal troubles where there are marked pigment changes. The differences are, however, pretty well marked. In chorio-retinal disease the pigment changes are represented by circumscribed plaques, which in addition to the black flecks show a discoloration of the entire surface. The shape of these spots is roundish, or, when they coalesce, of an irregular shape or ring-formed. Moreover, they lie behind the retinal vessels, and nearer or in the plane of the choroid, and the pigment does not follow the course of the vessels.

In retinitis pigmentosa, on the contrary, there is no real choroidal change, and even the pigment layer offers no conspicuous changes in itself. The configuration of the migrated pigment is stellate and not round, and the coloring matter lies along the vessels and oftentimes

on top of them. Even if in some cases this takes place in apparent choroidal disease, and retinitis pigmentosa is thus added to it, it is usually not difficult, although it may be so at times, to determine that the latter is a form of secondary degeneration of pigment, depending upon a pre-existing choroiditis or chorio-retinitis. When these are due to syphilis the commencement and course of the disease are usually different from that of the typical retinitis pigmentosa, for in the specific troubles the central vision is reduced and the peripheric limitations of the field trivial and not of a concentric form. The loss of vision, too, is much more rapidly progressive or one eye is much more, or exclusively affected. Here, too, as with other forms of syphilitic choroiditis and retinitis the iris is apt to be involved, and there are complications, such as paresis and paralysis of the external muscles and the accommodation.

Non-pigmented Sclerosis.—This form is a counterpart of the pigmented variety, with the remarkable exception that the changes in the pigment are, in general terms, wanting. It is not, however, meant by this that there are no traces of pigment degeneration, but that they are so trifling as to form no distinctive characteristic of the disease. It would seem as if, in the pigmented variety of sclerosis, that, there was a true hypertrophy of the pigment in which more pigment was generated than was absorbed, while in the non-pigmented kind there was a certain form of degeneration of the pigment and pigment-cell, in which the absorption was faster than the production. This gives to the fundus a washed-out, macerated appearance, which is very peculiar and which resembles a low stage of diffuse retino-choroiditis with minute dots of pigment here and there, with isolated and faded patches which may be of considerable size.

In certain cases the changes visible to the mirror are very slight, and cases have been reported with concentric limitation of the field of vision and night-blindness in which there were no ophthalmoscopic changes at all. I should be inclined, however, not to look upon these as true cases of sclerosis of the retina, for, if all other changes were wanting, the disease ought to manifest itself by a reduction in the diameter of the vessels. Moreover, it must be borne in mind that sclerosis of the retina may be the result of a variety of diseases, such as exudative choroiditis, chronic irido-choroiditis, and secondary glaucoma, and also that it may follow injuries to the cornea and sclera and other forms of inflammation.

Fine Strings in the Retina.—There is a peculiar kind of retinitis which is accompanied by the formation of fine white lines in the outer layers of the retina; and that these really lie in the outermost

layers is evident from the fact that not only do the larger vessels pass over them unimpeded in their course, but are clearly in a plane considerably anterior to them. Moreover, there are, at times, evidences of disturbances in the epithelial layer, or, at least, collections of pigment into greater or lesser masses in and around the starting or ending points of these formations. Jaeger gives two chromo-lithographic drawings of these "retinal strings," in one of which (Fig. 73) is an accompanying diffuse retinitis or, rather, neuro-retinitis of the inner as well as of the outer layers ("Hand Atlas," Taf. xvi., Figs. 73, 74).

It looks, at first sight, as if these bands must have some connection with the vessels, as they have often a branching and twig-like conformation; but a closer examination shows this to be erroneous, and that they are simply filamentous white or yellowish-white bands which, from their appearance, are strongly suggestive of hypertrophied connective-tissue elements, and that such is sometimes the case can not be doubted. Still, the fact that in one of the cases reported by Jaeger (Fig. 73) these strings entirely disappeared after a few weeks' duration would militate against such a view, and would be in favor of the presence of a less lasting product, and points to some temporary change in the tissue, which was capable of a complete absorption. I have seen several of these cases. One occurred in a young man who had a mild form of diffuse retinitis in both eyes, with the above-described appearances in the left eye. Unfortunately, the patient disappeared from observation after a few weeks, and the future progress of the disease could not be followed.

Septic Retinitis (*Embolic Panophthalmitis*).—It has been known for many years that in pyæmic conditions, the eye participated in the general disturbance, and that this was shown by a sudden and considerable swelling of the conjunctiva, pain in the bulb, and rapid loss of sight. These symptoms were accompanied by a disturbed condition of the media, with a yellow reflex from the fundus, followed by a *bombé* appearance of the iris with narrowing of the anterior chamber, which was often the seat of purulent collections—in fact, with all the evidences of a deep-seated and purulent choroiditis, iritis, and retinitis, which was followed in a shorter or longer time by phthisis bulbi, either with or without perforation of the globe.

This panophthalmitis was first observed by some of the oldest writers, in puerperal conditions, but was subsequently noticed as an accompaniment of or as a sequel to other disorders of a pyogenic character. The starting-point of the disease, so far as the eye was concerned, was at that time believed to be in the choroid, which was at first infil-

trated with pus, and from which the disease passed rapidly to the vitreous, retina, choroid, and other parts of the eye, leading to complete destruction of the organ.

Later it was shown by Bowman and others that there were cases of rheumatic origin in which there were no signs of pyæmia, or any purulent collection in any of the other parts of the body, but where, however, the autopsy showed signs of inflammation, with perforation of the aortic valves, together with the signs of fresh ulcerating aortic and mitral endocarditis, and purulent meningitis, without any essential change in the lungs or abdominal organs.

The true explanation of the nature of the disease was first shown by Virchow, in his discovery of capillary embolism, produced by particles of the inflamed and necrosed valves of the heart. Virchow showed that these particles after entering the blood were carried to the smaller vessels and capillaries of the body where they produced inflammations, and that not only in rheumatism but also in puerperal conditions this was brought about by ulcerating endocarditis.

It was further shown that for the eye, these capillary embolisms could occur in different membranes and in different parts of the same membrane, and that sometimes the choroidal and sometimes the retinal vessels, or both, could be affected at the same moment. The consecutive inflammation was consequently not always a choroiditis, but might be primarily a retinitis. It always, however, progressed very rapidly, and ended in panophthalmitis.

The embolic plugs in these cases consist of finely divided, granular or homogeneous masses, which often fill up completely great regions of capillaries, and agree in their constitution with the deposits upon the endocardium.

It was finally shown that purulent ophthalmitis could occur where there was no disease of the valves. This fact led Roth to observe, in an admirable paper, that all the cases of panophthalmitis could not be referred to capillary embolisms derived from the heart.* More lately still the belief has gained ground that these cases are as a rule due to the existence of micrococcal plugs, as masses of micrococci have been discovered by several observers in the vessels of the eye.

The affection usually occurs in a single eye, though occasionally both eyes are the seat of the trouble. In this category must be placed the metastatic choroiditis or ophthalmitis, which accompanies or follows an attack of cerebro-spinal meningitis.

In these cases the disease is manifested by a conspicuous yellow

* "Ueber Netzhaüt Affectionen bei Wundfieber."—Deutsche Zeitschrift der Chirurgie, 1872, vol. i, p. 471.

reflex, which usually occupies the entire pupil, and which becomes very brilliant under movements of the eye. When this exudation lies in the anterior parts of the eye and close to the lens, it is best seen with oblique illumination, as the anterior chamber is usually almost obliterated, the iris being *bombé* forward and the pupil somewhat obliterated and changed in shape. The surface of the exudation then appears to follow the curves of the lens and to be concave from before backward, and the mass as a whole has a soft, pultaceous look. The proper vessels of the retina are not visible, as they are entirely concealed by the exudation, but blurred red bands can sometimes be detected beneath the surface, while more rarely still actual vessels can be observed, which from their course and appearance seem to be vessels of new formation.

Under these conditions the mass has a very strong resemblance to a gliomatous growth and may be—as indeed has often been the case, in the hands of the unwary—taken for one. The chief diagnostic marks here, however, are the peculiar appearances of the anterior chamber, the *bombé* iris, and irregular shape of the pupil, which at times is almost quadrate.

Sometimes, though very rarely, in these cases of purulent ophthalmitis, the exudation is so thinly spread out over the surface of the retina that it can be seen, at least in its very earliest stages with the mirror better than in any other way. The exudation then has the appearance of a yellowish surface, which extends round the anterior parts of the field in a zonular form, and which comes into view in the extreme anterior parts of the eye.

Purulent infiltration into the vitreous gives at times a somewhat similar picture; but here, as a rule, no reflex whatever is obtained with the mirror, the pupil presenting a uniform blackness.

It may seem a little curious to the reader that I should be led into even this short discussion of a trouble which from its very nature seldom shows itself to the ophthalmoscope. The reason of this, however, is simply because in the graver forms the explosion of the disease is so sudden and so violent, that attention is first called to the eye when the media are so untransparent that an ophthalmoscopic examination is no longer possible. There can be no question, however, but that the disease must have a period of development, in the early stages of which signs would be manifest to the ophthalmoscope; and this must be particularly true in the case of puerperal women. All this is another reason why the ophthalmoscope should be used in a routine manner in every case of pregnancy, both before and after confinement. To the question of what use would it be, where little

or nothing can be done with our present knowledge to arrest the disease, I would say that the first step in gaining a knowledge of a disease, and the remedies to prevent it, is a knowledge of its presence, and the conditions under which it occurs. It might even happen, if the mirror was used habitually by general physicians as a means of diagnosis, that an ulcerative endocarditis of a mild form might be detected long before it was suspected by an exploration of the chest.

Besides the more severe forms, just alluded to, Roth, Litten, and others have described some milder forms of the same affection in the fundus of the eye, as the results of a pyæmic condition, of which those of the retina appear of great interest. An ophthalmoscopic examination of such forms, then, shows that small white dots occur in the region of the yellow spot, which are usually situated on both sides of the fovea. To these are added spots of ecchymosis, which, according to Virchow, are the constant accompaniment of stoppage in the retinal vessels. These ecchymotic spots consist of a collection of red blood-corpuscles, while the white flecks are composed of groups of thickened nerve-fibres connective-tissue cells, partly in a condition of fatty degeneration, and fatty degenerated capillaries, such as are seen in Bright's disease and other similar troubles. There is also at times a diffuse cloudiness of the retina.

This form of retinitis, which may be called retinitis septica, is distinguished from the more violent forms (embolic panophthalmitis) by being as much more frequent in occurrence, as it is milder in form. It is also characterized by the fact that the spots or masses in the retina are very small, circumscribed, and of a benign nature, with little or no tendency toward diffusion into the neighboring tissue or amalgamation with each other. The choroid is seldom implicated through hyperæmia, or by disturbance of the epithelial layer. The vitreous also shows but seldom any participation in the disease, either by an increase of its cells or other disturbances, though these may occur at times. This favorable course of the disease is due to the fact that this form of retinitis does not depend upon an embolic process, as in none of the cases which have been examined were there any stoppages of the retinal vessels found. The capillaries in the white flecks are either normal or show simple fatty degeneration, but contain no abnormal contents. The valves of the heart are also free from deposits (Roth, *loc. cit.*, p. 475). Kahler, however, found micrococcal plugs. According to Leber, lymph-corpuscles lie along the course of the vessels, showing that the process begins with them. According to Litten, hæmorrhages are the most common and significant symptom, at least in the more pronounced and purulent forms.

In the milder forms they also frequently occur, but to a less degree. They usually lie in close proximity to the vessels, especially near the veins—as, indeed, is commonly the case with all hæmorrhages. One great feature is the fact that these hæmorrhages begin to grow paler at the centres, under absorption, and not at the borders of the clot; and this sometimes begins to take place almost immediately after the extravasation has occurred.

Septic retinitis has been met with in senile gangrene, in double epidydimitis, with metastatic foci in the lungs and kidneys, in ulceration of the limbs, in venous thrombosis after operation, and, in fact, it may occur in any condition of a pyogenic nature.

While the occurrence of hæmorrhage into the retina is not symptomatic of any disease, still, the fact of its existence and the peculiar character which it is apt to assume in disordered states of the blood, and especially in pyæmic conditions, seem to point out at times, or at least to emphasize, the diagnosis as between these and some non-malignant troubles, such as typhus fever, and other diseases of a definite type.

Retinitis Proliferans.—The connective tissue of the retina is subject to a proliferation which may show itself in different places either by shred-like or membranous formations. That these feathery and membranous products may extend into the substance of the retina is shown by the fact that they often pass under the larger vessels on their way to the outer layers of the retina as far even as the pigmented epithelium. On the other hand, they often pass over the larger vessels toward and into the vitreous. This proliferating tissue may take its origin from the adventitia of either the larger or smaller vessels, from the radial fibres or from the limitans. The place of predilection would, however, appear to be the adventitia of the larger vessels upon and around the disk which they sometimes partially or even totally cover up with a dense white membranous expansion, which then usually extends along the course of the larger vessels for a considerable distance, as shown in the drawing (Fig. 42), which is from Leber.*

Manz† gave to this formation of connective tissue, not unjustly it would seem, the name of retinitis proliferans. Leber, however, doubts Manz's explanation of the nature of these formations, and attributes them to repeated hæmorrhages, which little by little take on a kind of organization. That repeated hæmorrhages into the substance of the retina or upon its surface do finally produce membra-

* Graefe und Saemisch, vol. v., p. 666.

† "Archiv für Ophth.," B. XXII., ab. iii., p. 229.

nous formations of a certain kind there is but little doubt in the mind of the writer, as he has seen such gradually formed under his own eye. These formations do not have the same look as those under dis-

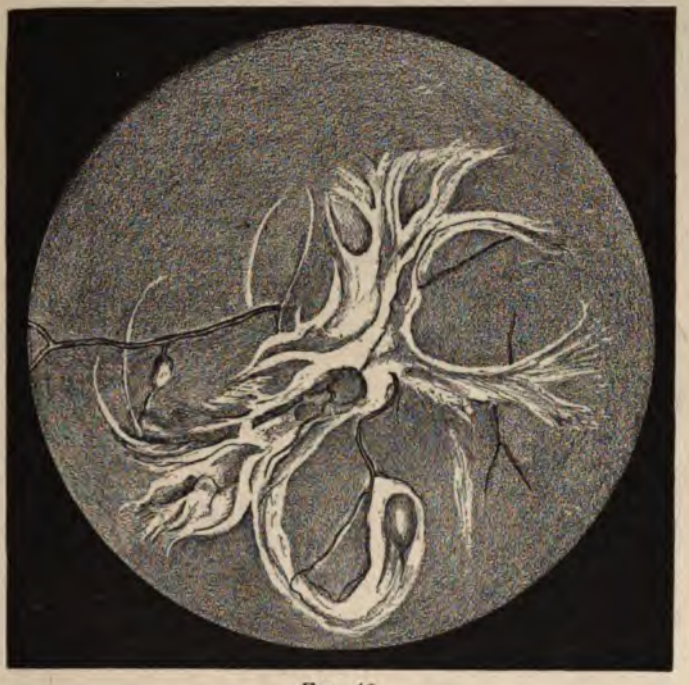

FIG. 42.

cussion, as they lack the laminated or honey-combed appearance of connective tissue, and possess instead a dull, yellowish aspect which more closely resembles an exudation, but which is evidently not one. It also at times looks as if the retinal tissue itself had changed in the neighborhood of these repeated hæmorrhages, and I have known such formations taken for superficial separations of the retina itself. The chief differences here are, however, that the new formation has, as a rule, no vessels upon it, and that the field of vision, though dulled at this place, is not entirely lost.

These membranes sometimes arise as the result of retinitis or neuritis, or indeed from choked disk, or they may occur where there are few or no visible signs of inflammation. Indeed, I have seen cases where there was good reason to believe that the formation was either congenital or acquired in very early life. Thus, I have seen a case where the entire optic disk was concealed by a rather dense membrane which extended for a considerable distance, after it had left the

papilla, along the vessels. At the porus opticus the vessels could just be discerned through the whitish covering, which produced in a striking manner the effect of a rather violent form of neuro-retinitis. The trouble was discovered merely by accident, as no complaint was ever made as to any lack of vision, which, indeed, amounted to $\frac{3}{8}$. Curiously enough, in these cases the sight is, as a rule, disproportionately good for the conspicuous ophthalmoscopic picture pre-

FIG. 43.

sented. Fig. 43 and Fig. 44 are from the article by Manz, and show very clearly the peculiar appearances that are characteristic of this disease.

Sometimes these membranes have but very slight attachments to the retina, and then appear rather as a disease of the vitreous, in which they seem to take their origin. Careful examination will, however, often enable one to trace their attachments up to the retina. They sometimes become vascularized, and then present a most beau-

tiful appearance. They have already been described in Vol. I., page 177, *et al.*

Separation of the Retina.—The principal cause of separation of the retina is the collection of a serous fluid between it and the choroid, from whatever reason it may have been produced. This sub-retinal fluid is usually clear and of a yellowish color, although at times, when there has been extravasation, of a reddish tinge, which is due to the

FIG. 44.

presence of hæmatin from the suspended blood-corpuscles. It is commonly very rich in albumen and is easily coagulated. In separation of the vitreous the fluid which lies between it and the retina is of the same nature.

Separation of the retina usually shows itself to the mirror in a very palpable manner by the presence of a larger or smaller grayish undulating fold or protrusion which occupies different parts of the field of the pupil under excursions of the observed eye. It is most

easily detected by simple observation with the mirror from a distance, but closer inspection with the proper correcting glass usually shows the protruding surface more in detail. This is then found to consist of grayish folds or furrows which run generally in an equatorial but sometimes in a meridional direction. These folds are of a lighter color on the summits and of a deeper shade in the depressions than the rest of the surface, on account of the difference in reflection both as to quantity and direction of the light, thus giving the appearance oftentimes of light bands running over the surface of the elevated retina. The grayish color of the separated portion is due in part to changes in the membrane itself, in part to the angle at which the light is reflected, in part to the color of the fluid behind it, and, finally, in part to the height of the separation.

Separations of the retina may be either partial or total. When total, they offer but little for inspection by the mirror, for then the retina is usually gathered into a funnel-like cord which completely blocks the pupil, and is better seen under oblique illumination than with the mirror. If partial, it may vary from very minute detachments of a millimetre or two in breadth, described by Knapp, to what includes the greater part of the retina. Leber mentions having seen with the microscope minute separations of the retina near the optic nerve, especially in neuro-retinitis, and Gower gives the ophthalmoscopic appearances of a peculiar spot in the retina, which he thought must be due to one of these small separations, as one was brought to light upon section of the eye. I have myself been able to detect very small, almost filamentous detachments of the retina, but have never seen those near the optic nerve, described as occurring in neuritis at the autopsy. These are probably overlooked, because the tissue of the retina overlying them is too opaque to render them visible with the mirror. We occasionally see a number of small separations in the region of the macula, which are then arranged in narrow folds which sometimes assume the appearance of the furrows seen on a beach after a receding tide.

The amount of motion which the separated portion of the retina undergoes under movements of the observed eye varies with the height of the separation, the character of the fluid behind it, and especially the consistency of the vitreous in front of it. The more liquid this is, the greater and the quicker the movement, and the more wave-like and tumultuous the motion.

When the detachment is of long standing, and there have been changes either inflammatory or retrogressive going on for some time,

the retina has a deep gray tinge, which is no doubt much increased when the fluid behind it contains, as it not infrequently does, diffuse or concrete opacities which naturally reduce its transparency. There are at times peculiar bands of a yellowish color which run across the separated portion of the retina, but which are evidently below the plain of the larger vessels and in the outer layers of the retina, the precise seat of which it is difficult, however, to determine, as well as their nature, they being considered as due by some to coagulated albumen on the surface of the fluid behind the membrane. At times they bear a resemblance to the so-called "retinal strings," which have already been described as occurring in the substance of the retina where no separation has taken place.

In some cases the separated portion becomes so untransparent as to reflect large quantities of light, so that it acquires even a whitish hue, especially in places where the appearance is given of white flecks of greater or lesser size, which sometimes have a glittering aspect, as if due to fatty degeneration. Sometimes, also, minute specks of cholesterine are present on the surface of the retina, or, in rare cases, in the effusion behind the membrane; chalky deposits may also occur at times in the separated portion of the retina. Sometimes, on the contrary, the separated part of the retina, instead of having a grayish color, preserves to a great degree, or almost entirely, its transparency, and the effect produced then depends upon the nature of the fluid behind it. If this has remained clear, then the details of the choroid can be seen with comparative clearness. Under these conditions it is sometimes difficult to detect the presence of a separation, or, when the elevation is detected, to say whether this is due to a detachment or to a morbid growth—a difficulty which is increased when there happens to be little or no motion in the separated portion; and especially is this true when the separation is in the anterior part of the eye, and close to the ora serrata. In this case the vessels, which are naturally small, are rendered to the eye still more so by being so far within the focus of the dioptric system, and as a consequence, are so blurred and indistinct, as not to be discernible except with a strong correcting glass. For this reason it is better, when a separation is suspected, to make an exhaustive examination under a dilated pupil, both with the inverted and upright image, with correcting glasses of varying and increasing strengths.

If, moreover, it should happen that the fluid behind the retina is semi-transparent, the resemblance to tumors of a gliomatous nature is heightened, and double care is required not to make an incorrect

diagnosis, under which an enucleation might be advised and performed, as has, indeed, happened before now.*

In separation of the retina the vessels appear broken and displaced sidewise, as they mount over the elevated portion, and have a different aspect both as to their course and color. They appear somewhat smaller, as they lie inside of the focus of the dioptric system, and seem to have lost their arborescent distribution, and to run like dark-red lines, which are unevenly stretched out. Sometimes, however, not only is the normal amount of tortuosity preserved, but it is increased.

The color of the blood-column is, as a rule, darker than normal, both in the arteries and veins, and the impression is gained that this darkness in color is due to some impediment in the circulation. That this, however, is not so, is shown from the fact that the blood has its usual color at the place of transition of the vessel from the non-separated to the separated portion. The darker color is really due to the fact that the vessels are seen in part by transmitted light, which oftentimes, when the retina has preserved its transparency, predominates over that reflected from its surface. It is important to notice this increased darkness of the blood-column, as it is often through this that the separation is first detected and a knowledge formed of its extent—that is, in its earliest stages, and before a protrusion to any degree has occurred. The increased darkness is also at times of service in determining whether an advanced position of the retina is caused by a fluid substance behind it or a solid growth, especially in the early stages of tumor, for in the latter case the vessels do not appear of a darker shade, but are even at times of a brighter and more scarlet hue.

This diagnostic mark, of increased darkness of the blood-column, may be supplemented by another which consists in the absence of the light-streak, which often leads to the detection of a commencing separation in its very earliest stage—probably, indeed, when no actual separation has taken place, but when only the merest film of fluid has inserted itself between the choroid and the rods and cones. As an example of this I would say that I once observed a case where there was a sudden loss of nearly half the visual field, and where, upon the most careful examination, no elevation could be detected. Over that portion of the retina, however, which corresponded to the defect in the field the light-streak was entirely absent, leaving off abruptly at the borders of the part which did not functionate. There were absolutely no other ophthalmoscopic signs. This condition con-

* Case of supposed glioma, "Trans. Amer. Ophth. Soc.," 1869, p. 70.

tinued for something over a week, when actual separation occurred, which was precipitated by long-continued stooping in weeding in a garden on a very hot day.

There is oftentimes, also, a parallactic displacement to be obtained between one part of a vessel upon the elevated portion, and another part of the same vessel upon the portion of the retina which is still attached; or between a vessel of the retina and one of the choroidal vessels when the fluid behind the retina is sufficiently transparent.

Sometimes the place of juncture of the separated and non-separated portion is surrounded by a yellowish ring which has the appearance of an exudation, and which suggests the idea that an effort is being made to stop the extension of the separation by forming a point of union with the underlying membranes. This may at times also be the seat of a greater or less accumulation of pigment, which may go on increasing until a ring is formed.

In partial separation of the retina, those portions which are not separated show but little change. There is, as a rule, but little or no injection of the optic nerve, and little or no hyperæmia or tortuosity of the vessels, and but few signs of a diffuse retinitis. Indeed, oftentimes the non-separated part has a perfectly normal appearance.

According to some authors, spontaneous rupture of the separated portion can occur, and the ragged edges of the opening which has been formed can be seen, while the details of the choroid come clearly into view. Noyes has reported a peculiar form of separation which involved the macula lutea in such a way that the peripheric portion was torn away from its attachments in a circular manner while the central portion remained fixed and in its normal plane. The effect produced was as if the separated portion had been cut with a punch. It will be remembered that the writer has described seeing cases of central retinitis where there was a solid elevation of the retinal tissue, and where there was the same effect of being cut with a punch (Vol. II., p. 138).

Separations of the retina that are caused by hæmorrhage behind the membrane, and which can be seen, are, in the writer's experience, very rare. These usually occur after injuries, and are accompanied by so extensive an obscuration of the media as to prevent an illumination of the fundus—at times, indeed, to such a degree that no reflex at all is obtained. Sometimes, however, such separations do occur of a limited extent, especially in the more anterior parts of the eye, and for some reason or other in the upper part. With the ophthalmoscope such separations appear black, and it is only at the edges and under the most favorable incidence and reflection of the light that any red tinge is perceptible. This, however, can sometimes be pro-

duced, when all other means fail, with oblique illumination with a dilated pupil, when the separation lies in the anterior parts of the eye.

Spontaneous Reattachment of the Retina.—This may take place after a longer or shorter interval. Should this be short and but little change have taken place, the reattachment may occur and leave but few or no visible signs—so far as the mirror is concerned—that any detachment has taken place. On the other hand, when changes have occurred in the epithelial layer and choroid, these remain, and show themselves usually in linear or circumscribed discolorations and displacements of the pigment, with yellowish-red spots and streaks, which are evidently in the pigment layer or in the choroid or in both.

Commotio Retinæ.—Injuries to the head, such as concussions and blows upon the eye itself or its surroundings, especially from blunt instruments, may express themselves in such a diversity of ways and in so many different parts of the eye as to include almost every phase of injury; as the cornea, sclera, iris, lens, choroid, and retina, may be either singly or conjointly affected. So far as this last membrane is concerned, it may be stated in a broad and general way that the results of these injuries may be divided into two classes, those which produce appearances that are visible to the mirror and those which are not; for it can not be denied that there are cases, rare though they be, in which after such blows there is not, at the time at least, the slightest evidence of any damage done excepting a greater or lesser deterioration of the vision, which is usually proportionate to the degree of the violence and which may vary from the slightest veiling of objects, to almost if not quite a total loss of sight.

It must be admitted, however, that in many if not in most of these cases in which there are no ophthalmoscopic signs at first, such signs do occur if the opportunity is given to observe them for a sufficient length of time. When this is possible, the signs of white atrophy of the optic nerve usually become evident. These may be accompanied not only by a diminution of the vessels, but also by some very slight but still evident manifestation of a mild form of vasculitis, in which the light-streak becomes decreased or invisible, the vessels lose their tapering aspect, and are sometimes accompanied upon and near the disk with fine white lines. There may be, too, a want of definition in the general fundus, with barely perceptible changes in the choroid and pigment layer—such an appearance, indeed, as is sometimes seen after a post-ocular neuritis, similar to what is supposed to follow the abuse of alcohol or tobacco. All this suggests the idea that the force of the blow has propagated itself through the eye or by means of the bones of the orbit and created on the mechanical principles of

contre-coup some shock to the nerve or even brain, which expresses itself in some molecular change in the nerve itself without producing retinal changes, and which may be accompanied with or followed by some of the evidences of a post-ocular neuritis and secondary atrophy.

But to return to the milder forms. When a blow of moderate force from a blunted instrument, such as a stick of wood or a cork expelled from a soda or champagne bottle, strikes directly upon the eye, it produces the following train of symptoms almost immediately after the accident: In the typical cases there is usually a slight scleral injection around the margin of the cornea, or this may be entirely wanting. Occasionally, however, this injection is more marked, and may even extend over the entire white of the eye, such as we see in mild forms of iritis. The iris is usually somewhat contracted, sometimes markedly so, especially in young people, and obstinately resists even repeated instillations of atropine, or, when it does yield, does so in an irregular shape. There may be also some discoloration of the iris with the other signs as mentioned above of a low form of iritis or irido-choroiditis.

The media are usually clear, and the first thing that attracts the attention of the observer is the remarkable appearance which the retina exhibits. Usually within an hour or two there is in the neighborhood of the disk, or at the macula, a grayish circuit of greater or lesser extent and which varies from a spot as large as the disk to one which may embrace the greater part of the outer portion of the fundus. Closer inspection shows that these larger districts are made up of smaller ones, between the borders of which the dimmed but still rosy-red of the background of the eye shows through. After the first day, or perhaps two, the grayish color becomes lighter but more dense in character, until the smaller patches coalesce into one uniform mass of a very light gray or at times even milk-white color, such as is sometimes seen in embolism.

It would seem as if these patches of a gray and white color must be due to an acute œdema into the retina, as is shown by the rapidity with which they appear and are reabsorbed. Berlin observed a case in which there appeared to be two foci of infiltration—one situated in the anterior part of the eye, where the force of the blow was first experienced, and the other directly opposite, and he is of the opinion that this occurrence would be observed oftener if the point of contact of the blow was not so far forward as to be beyond the reach of the ophthalmoscope.

It is commonly stated that the vessels are not, as a rule, much

affected, but in the cases which I have myself seen there has usually been a perceptible reduction in the size of the arteries and a corresponding enlargement in the veins, which are usually tapering toward the porus opticus. This effect was very conspicuous in one which has already been mentioned, where there was a marked injection of the sclera, discoloration of the iris, a reduction in the size of the arteries, and a marked increase in that of the veins. There was also reduction in the tension of the eyeball. The effect was supposed to be due to some vaso-motor disturbance of a neuro-paralytic origin, the result of the blow (see Vol. II., p. 9).

The vessels usually run unhindered over the grayish-white infiltration, which shows that it must be either in the middle or outer layers, or those in which the capillaries are situated, or at least in those layers exterior to the fibre layer in which the larger vessels lie. At times small hæmorrhages have been observed near the vessels. These I have never seen, but I did on one occasion observe a very large hæmorrhage in the anterior parts of the eye, that was evidently of choroidal origin, and between the choroid and the sclera. There had, however, been penetration of the eyeball in this case, which always favors the production of hæmorrhage.

After a day or two, or when the infiltration has assumed the white appearance alluded to, it begins to disappear, decreasing in extent and becoming again of a grayish color. It disappears entirely, as a rule, within two or three days. The effect, however, upon the vision lasts much longer, and seems, as Berlin has pointed out, not to be dependent upon the amount of the infiltration or its position at the macula, for the sight begins to improve slowly before the infiltration reaches its height, but is not entirely re-established until long after it has disappeared.

When the blow has been directly upon the eye itself, and not of great force, the disturbance in vision is ordinarily much less. Berlin, who has studied this form of retinal disease very carefully,* has also remarked that in these cases the slight reduction in vision may be due to the production of some form of irregular astigmatism, and that this is then probably seated in the lens. In corroboration of this, I would say that I have observed several cases where, upon careful examination, a sort of folding in the anterior capsule of the lens was plainly observed, and which came out very conspicuously by the use of a strong correcting glass under slight rotations of the mirror. In these cases there were absolutely no changes in the back of the eye. This ruffled appearance of the anterior capsule faded away after

* Zehender, 1873, p. 42.

a greater or lesser period of time, when vision returned to the normal degree. I have also seen a ruffling of the membrane of Desemet as the result of a blow.

McHardy reports a case of double-sided retinitis which resembled that of Bright's disease, and which occurred after an injury to the back of the head.*

Berlin has suggested the name of amblyopia traumatica as more appropriate than that of commotio retinæ, while, for the milder forms, in which there are no ophthalmoscopic signs in the retina, Leber proposes the term of traumatic anæsthesia.

Rupture of the Retina.—Pure rupture of the retina, of the kind that occurs so frequently with the choroid from blows or injuries, is exceedingly rare. I have seen but one example of it myself. This happened in the case of a gentleman who, while riding, was thrown from his horse, striking upon his head. A large rent in the retina was produced, occupying nearly the entire posterior pole of the eye, including the region of the yellow spot, which allowed the stroma of the choroid to be seen through the opening. There was no sign of any rupture of the choroid in the exposed surface. The ragged edges of the retina could be plainly seen projecting into the vitreous, while the lower part of the retina, from the edge of the rent, was separated toward the equator.

Cysts of the Retina.—Under the name of œdema of the retina Iwanoff described a condition of the retina which had been observed before, but not so accurately studied. For the title of œdema of the retina, Nettleship proposed perhaps the more appropriate name of cystoid degeneration. As a matter of fact, according to Leber, the disease would seem to be due to the presence of œdema combined with that of cystoid formations. It consists of a collection of fluid in the two granular layers, which, gradually increasing in quantity, may acquire an enormous size. It occurs as a senile change in elderly people, and, in this case, always in the anterior parts of the retina near the ora serrata, and presumably beyond the reach of observation, as no case of it has yet been detected in life. As Iwanoff showed, however, it may occur in other places in the retina and in younger people.

As cystic formations are very rare, and as so far no single example of it has yet been diagnosticated with the ophthalmoscope, the following case, believed to be unique, will be of interest. The case occurred in a woman of middle age in the practice of my colleague, Dr. C. Cocks, and was sent to me as a peculiar and intense form of retinitis,

* "Trans. Ophth. Soc. United Kingdom," vol. ii., p. 54.

with a transparent separation of the retina. The retinitis, with but slight changes, had been present for the past two years. The separation, however, was of a very recent date.

The fundus presented all the appearances of a pronounced form of retinitis, with plaques of exudation, great vascular disturbances, with signs of perivasculitis and hæmorrhage, all of which, however, were limited to the upper and outer portion of the fundus, the rest being normal. In addition to these signs there appeared to be a well-marked detachment of considerable size and prominence of the upper and outer part of the retina. The separated portion, or what was supposed to be such, was almost perfectly transparent, while the fluid behind seemed to be of a yellowish color, and to possess a good deal of transparency, but was not sufficiently clear to allow the details of the choroid to be seen. The surface of the elevation was smooth, and covered at intervals with fine lines, and of a color which varied only a little from the transparent membrane itself. Closer inspection showed that these lines were not vessels at all, but were of the same nature as the rest of the membrane, and were apparently re-enforcements of the same substance. They were arranged so as to form a sort of network, and gave to the surface of the projecting body a frosted appearance, such as is sometimes seen on the surface of a transparent Japanese vase.

It was decided, after a long and careful study, that what was at first taken for a separation of the retina was in reality a cyst filled with a semi-transparent fluid.

When the patient returned again in the following week, the entire protuberance had disappeared without leaving a trace of its existence which could be identified with any accuracy. Signs of inflammation there were in abundance, but no trace whatever of any rupture or the resulting scar; and I am informed by Dr. Cocks that such is the case at the present day, when some eight months have elapsed. The other eye showed no signs of disease.

The discovery of small cysts on section of injured and diseased eyes, though rare, is still not a very infrequent occurrence, but, as they can not be detected with the mirror, they do not concern us here.

Dazzling of the Retina.—Under certain circumstances, prolonged exposure to direct sunlight, as in the tropics, or to intense reflection from the snow, or the glare of the sea, produces a form of diffuse retinitis or neuro-retinitis. Much more frequently, however, such exposure is followed by amblyopia which, as a rule, is not accompanied by any ophthalmoscopic signs. These affections, such as night-blindness, moon-blindness, and snow-blindness, are then more akin to

what is known as fatigue, or an exaggerated torpor of the retina, and are probably due to some derangement in the normal secretion of the visual purple, since confinement in the dark, with consequent increase of the purple, usually restores the sensitiveness of the retina for reduced illumination.

After-images of the sun, such as may occur from prolonged looking at an eclipse, often produce a central scotoma, which may be temporary in its character, persist for some time, and then disappear, or remain permanently. I have seen several such cases as the result of an eclipse, and one or two after an exposure to an intense light of a lamp or a fire. I have never been able, except in one case, to detect any ophthalmoscopic signs. In this case there was a very delicate and gauze-like opacity at the macula in one eye, with slight disturbances in the pigment layer.

On one occasion I examined the case of a young lady who had looked intently at the sun during the transit of Venus through a binocular opera-glass. In this case both eyes were affected, there being in each a small central scotoma, which cast a veil over all objects looked at. Dr. Mathewson, who saw the case immediately after the exposure, informs me that vision was reduced in each eye to $\frac{2}{7}$. When I saw the patient, vision had risen to $\frac{3}{4}$, where it has remained to the present day. There were no ophthalmoscopic signs, either immediately after the exposure or subsequently. Dr. Mathewson also reports that another member of the family was affected in the same way with a double central scotoma, which after a short time entirely disappeared.

Cases of retinitis have also been reported as the result of a single intense flash of light.

CHAPTER V.

DISEASES OF THE OPTIC NERVE.

For the purposes of ophthalmoscopy the head of the optic nerve may be divided into three parts: (1st.) That which is opposite or in a plane with the scleral opening. (2d.) That which lies opposite the choroid. (3d.) That which lies opposite the retina, including the nerve-fibre layer—i. e., the papilla. (See also Vol. I., page 27, *et al.*)

The retinal portion is supplied by the vessels of the central system, the choroidal by those which come from the choroid, and the scleral those which come from the scleral ring, which, it will be remembered, is a vascular circle situated in the tissue of the sclera and which surrounds the stem of the nerve as it passes through the scleral opening (Vol. I., page 30). It will be understood, of course, that this division is not a rigid one, and that vessels from one part pass to the neighboring part, so that there is a connection between the vessels from the retinal on one side and the choroidal on the other, and to a less degree between the choroidal and scleral portions.

Nevertheless, under the effects of irritation and inflammation a more definite demarkation takes place than in health, so that a distinction between a retinal and choroidal congestion can be made with a good deal of certainty, which is also applicable, although to a less degree, to a differentiation between choroidal and scleral injection.

Irritation of the Optic Nerve.—When the increased injection is in the anterior parts of the optic nerve—that is, in those which are supplied by the smaller and more anterior vessels of the central system—it is more radiating in character, following the direction of the nerve-fibres and the course of the larger vessels (Fig. 45, which is from Jaeger). As the congested vessels pass over the scleral ring they have a tendency to conceal, or, if the congestion is marked, to obliterate it, while the boundaries of the nerve at times become unrecognizable. This may occur in any eye in which for any reason, such as overwork or exposure to glare, there is retinal irritation. It is often seen in highly hypermetropic eyes to such a degree as to lead to the belief that actual inflammation is present, which finds its ex-

pression in a low form of neuritis. This increased vascularity is supposed to be due by most authors to the increased accommodative efforts required by the structure of the eye. Still, I can not help thinking that oftentimes the effect is increased through contrast, from the presence of an unusual amount of connective tissue which is common to such eyes and which brings out the striated appearance to a greater degree, helped, no doubt, by the fact that so much connective tissue requires perhaps a greater degree of vascularity for its nourishment. Thus a normal hypermetropic eye may seem to be congested, while it is not so in fact.

Fig. 45.

In choroidal irritations and inflammations, on the contrary, we usually see that the injection is not so striated, and presents a more uniform appearance and color and is deeper situated (see Jaeger, Figs. 102, 105, 106, 108–126). It is also more liable to affect the central parts of the nerve, leaving the connective-tissue ring more clearly defined from contrast than in the normal eye. At least it does so in those cases where the more posterior vessels are affected, and where, as I believe is often the case, the inner sheath of the nerve is continued farther on in its course. This effect would be still more increased if the choroidal ring itself happened to be unduly developed. This effect is due to the fact that, in these cases, the congestion comes from the vessels of the choroid and scleral ring, which lie on a plane posterior to the connective-tissue ring, which there appears more conspicuous from contrast, which is also increased from the fact that the connective-tissue string (Vol. I., p. 25, Fig. 8) which surrounds the central vessels, and which in the normal eye is destitute of vessels, becomes vascularized.

Irritation of the Scleral Region.—When this region alone is affected the injection appears to lie still deeper, and is, as a rule, more

uniform in character; but there are cases where just the reverse takes place, and the injection has an interrupted or spotty character, which is due to the meshes and interspaces of the lamina cribrosa. This is very clearly seen in some cases of excavation of the optic nerve, due to a choroidal or glaucomatous process.

This scleral form of injection is often seen, according to Jaeger, in brain-disease, and I myself have frequently seen it in those who are victims to sick-headache.

The congestion which follows irritations of the nerve alone, or of the nerve and retina, can be and often is confined to the smaller vessels and capillaries, so that it is not only allowable but appropriate to speak of a capillary congestion independent of that of the larger vessels, which are usually not affected in any degree that is appreciable to the mirror.

When irritation of the nerve is joined to that of the retina, we simply have what is usually expressed under the title of retinal irritation, which means that the finer vessels extend over the connective-tissue ring into the retina.

Inflammation of the Optic Nerve.—Neuritis.—Just as it is useful to designate the vascular supply of the optic nerve by the region which it occupies, so it is convenient to divide the substance of the nerve into regions, and to designate the inflammatory condition by the name of the part inflamed; as, for example, "papillitis" to denote an inflammation of the papilla. The difficulty has been, that here, as elsewhere in medicine, names sprang up in the early days of ophthalmoscopy from the supposed nature or cause of the inflammation, which advancing science proved to be incorrect; and here, as elsewhere, the older names have been retained to a large degree, in spite of the fact that they did not represent the pathological condition to which they owed their title.

The nomenclature proposed by some of the more modern authors, notably by Leber, would on the whole seem to be the best, and this has been followed to a great degree in the present instance.

We divide, therefore, the principal forms of inflammation according to the part of the nerve which is affected. Beginning with the outside of the nerve, we have—(1st.) Peri-neuritis, which signifies that the seat of the inflammation is in the sheaths of the nerve. (2d.) Papillitis (choked disk), where the papilla alone is affected, or that portion of the nerve which is included between the lamina cribrosa and the anterior surface of the nerve-fibre layer. (3d.) Papillo-retinitis, where the retina is also affected. (4th.) Interstitial neuritis, where the connective-tissue stage-work of the nerve-stem is affected. (5th.)

Medullary neuritis, where the nerve-substance, or medulla itself, is affected. Furthermore, we speak of a partial neuritis, where a portion of the disk is seen to be inflamed; and a retro-bulbar, or post-ocular neuritis, where the disease is supposed to be posterior to the nerve entrance. Again, we speak of an ascending neuritis, where the inflammation is supposed to progress from the eye toward the brain, along the sheaths and stem of the nerve; and of a descending neuritis, where the inflammation descends from the brain toward the eye. The great majority of practitioners, however, neglecting these fine-drawn distinctions, use the terms neuritis and neuro-retinitis to express all forms of inflammation of the nerve and its surroundings, with the one great exception of the frequent application of the term "choked disk," rendered classical by Von Graefe, to that particular form of neuritis which is dependent upon brain-disease, and notably to tumor of the brain.

It will be understood, from what is stated above, that many if not most of these distinctions are applicable to the microscope and can not be differentiated by the mirror, except under the most favorable conditions—for this reason, if for no other, that they rarely exist alone, and, when they do, are sometimes situated in positions which are not visible to the observer.

Simple Neuritis.—Irritation of the nerve passes gradually into inflammation, so that in the earlier stages the difference between the two is rather one of degree than of kind. In the milder forms the smaller vessels and capillaries would appear only to be affected, the larger vessels not partaking in the disturbance to any appreciable degree. The injection of the nerve becomes more marked, while the smaller vessels, increasing in size, become visible in greater numbers. The disk, as a whole, has a streaked and fretful appearance, which is easy enough to detect, but difficult to describe, and which yet distinguishes the milder forms of neuritis from simple hyperæmia, or passive dilatation of the vessels (Fig. 46). This plate is taken from Gowers's "Medical Ophthalmoscopy," and represents in a very graphic manner the changes taking place in the different stages of disease of the optic nerve. Number 1 represents simple congestion of the disk from embolic softening, and is from a drawing of the inverted image, while number 2 is the same disk as seen by the direct method, though reduced. Numbers 3 and 4 are commencing optic neuritis, "congestion with œdema"; the former by the inverted image and the latter by the upright. Numbers 5 and 6 represent optic neuritis, probably from a cerebral tumor and are of the same eye, though seen first by the indirect and then by the direct method of

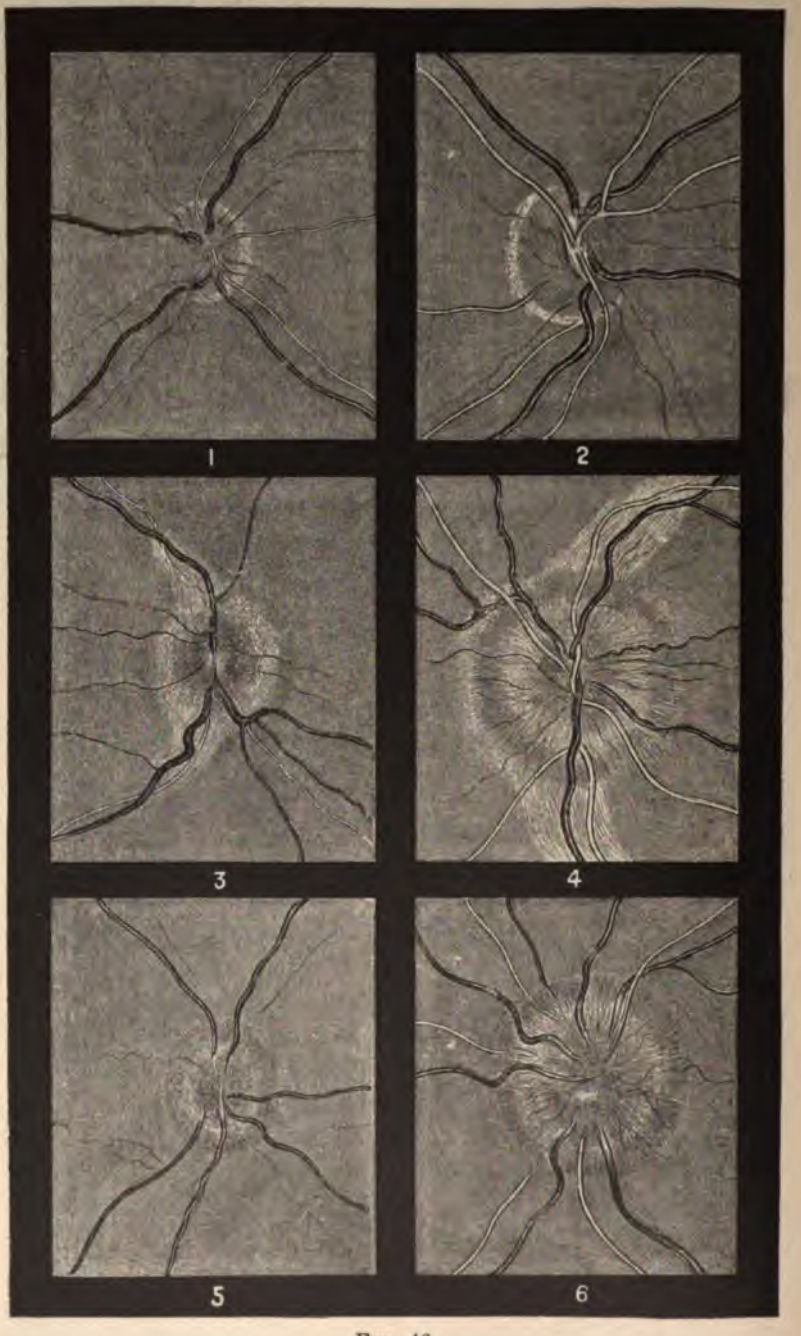

FIG. 46.

examination. This placing side by side of the two illustrations of the same eye gives a most admirable example of the results of the two methods and of the advantages that can be derived from each.

In the early stages of the more moderate forms, or even in the entire duration of the trouble, there is usually but little difference in the level of the disk, and commonly but little or no œdema of the tissues. The edges of the nerve are, however, somewhat obscured by the increased number of congested vessels, which pass over the connective-tissue ring to such a degree, in some cases, that the position of the disk can only be told by the convergence of the vessels. Occasionally, however, it would appear as if the inflammation, even in these milder forms, was of an œdematous character, and as if a light infiltration had taken place almost in the very commencement of the morbid action. Here the injection, instead of being increased, is lessened, and the whole surface of the nerve has a faded and cottony aspect.

The delicate and smoke-like cloud may extend out into the retina on all sides, or be limited to a certain portion shooting out from the borders of the disk in a comet-like fashion. This form of neuritis is apt to accompany syphilitic retinitis and chorio-retinitis, and is often also accompanied by changes in the vitreous, which adds to the faint and washed-out appearance of the nerve. I have seen this form of neuritis also with the milder forms of brain-disease where the trouble was limited to the meninges, and was not due to gross lesions, such as tumors or growths.

As the type of inflammation increases, the centre of the disk becomes redder than the periphery and is much more uniform in its coloring, and is less striated than at the circumference of the nerve, which, instead of being of a deeper red, may assume a grayish color, showing the presence of œdema. The physiological excavation fills up sometimes so completely and so rapidly as to lead to the impression that there was really no excavation ever present, and that what appeared as such at the porus was in reality tissue, which was so transparent as to be invisible to the mirror until it was rendered opaque by the inflammatory action. A step farther, and the contours of the nerve appear not only to be on the same level with the rest of the disk, but even slightly in advance of it, or the nasal half of the disk may be more swollen than the temporal, as the tissue in this place is more abundant. The œdema now becomes more marked and there may be some small hæmorrhages near the nerve, and the neuritis then passes from the milder forms to the graver and assumes the features of choked disk.

Graver Forms of Neuritis.—As has been seen, the milder forms of neuritis may pass step by step into the more serious forms; and this is especially true with those which are due to local intraocular processes in which the retina plays an equal or more prominent part, as, for example, the neuro-retinitis of Bright's disease. In these cases it would seem that the congestion in the earlier stages was due to the effect of the disease upon the smaller vessels, and that tissue changes, such as infiltrations and exudation, preceded any visible effect upon the larger vessels. It would seem, on the other hand, to be different with that form of papillitis or choked disk which is dependent upon some gross lesion of the brain. Here, as a rule, the first perceptible change is noticed in the larger vessels. This consists of an enlargement of the veins, which is ordinarily accompanied with increased tortuosity. The arteries, on the contrary, are reduced in caliber and run a straighter course. These two symptoms, which occur before any congestion of the disk is visible, may be carried to a considerable degree, so that it may be difficult to determine whether one is dealing with one of those rare cases of exaggerated tortuosity of the veins which occur in the normal eye or with a commencing choked disk. When due to a normal peculiarity, the arteries usually follow the same general curves of the veins.

The vessels also in the normal eye are almost invariably in the same plane as the surface of the retina, no matter how great is the tortuosity.

In papillitis (choked disk) we often have, however slight it may be, still a perceptible antero-posterior deviation, while the arteries, instead of following the curvatures of the veins, are even straighter than in the normal eye. In stasis hyperæmia, which is not inflammatory, it will be remembered that the arteries are enlarged and tortuous.

The enlargement and deeper color of the veins and diminution of the arteries, which are the initial steps in choked disk, may continue in certain cases even for weeks. Usually, however, the other steps follow each other with more celerity; sometimes, indeed, with fulminating rapidity. The injection of the nerve increases not only by the distention of those vessels which already existed, and which belong naturally to the part, but by—as the microscope teaches—vessels of actual new formation, so that quite early in the disease, as in other forms of neuritis, the nerve has a red and striated appearance, sometimes markedly so. The physiological excavation rapidly disappears, so that the surface of the disk is all on one plane. Then the head of the nerve begins to protrude beyond the surface of the retina, and this

still increasing gives to the head of the nerve a formation like that of a mushroom. Over this the central vessels run, and dip down to the level of the retina with a broken and disjointed appearance. Their tortuosity increases at this time, until they acquire that snake-like appearance which has given to the disk the appellation of the Medusa nerve.

The surrounding retina now begins to be implicated; the nerve loses all traces of its outline, and its position can often only be told by the convergence of the vessels toward the centre of the nerve, or toward what was its circumference in case the centre has become obliterated. The vessels then seem cut abruptly off at a considerable distance from the original borders of the nerve. Plaques of exudation, of greater or lesser size, appear upon the surface of the papilla and neighboring retina, with hæmorrhages more or less profuse in character. These are in the inner layers, and usually along the course of the larger vessels. Sometimes fringe-like hæmorrhages are arranged in a circle round the projecting head of the nerve, or mark the position where the vessels are lost to view in the swollen tissue as they converge toward the enlarged circumference of the nerve. The hæmorrhages may extend over the greater part of the retina, and sometimes far out toward the ora serrata. The arteries may disappear entirely, buried beneath the swelling tissue, and only here and there a distended and interrupted vein may make its appearance in what seems to be the general ruin. The swelling goes on increasing, both in a lateral and perpendicular direction, until the disk, if visible at all, seems to have twice its size. The perpendicular elevation may attain a height of two millimetres above the plane of the choroid. This can be plainly seen by the movements of the lens in the indirect method, or measured exactly with the upright image, as has already been pointed out in Vol. I., page 115. The swelling may be greater in one part of the nerve than the other. The edges of the nerve are sometimes so steep that they fairly seem to curl over into the retina, while at others they slope gradually away, the whole swelling forming a shallow plateau extending over a large circuit. The condition may now be fairly called a neuro-retinitis of a flagrant type, especially as there are hæmorrhages, plaques, and exudations extending far over the surface of the retina, concealing in part and in part reducing the tone of the general fundus.

Curiously enough, through all these changes the light-streak upon the vessels, where they are still visible, is preserved, oftentimes in all its brilliancy and increased in breadth, as would naturally be the case when the veins are dilated. This proves that the current is certainly

going on in that particular vessel, no matter how much its disjointed appearance may give the idea that it is strangulated.

It has just been said that the physiological excavation was usually filled up, and this is true as a rule, even in the earlier stages of the disease; but there are some exceptions to this rule, for, when perineuritis is present in a predominating degree, associated with circumpapillary retinitis, the physiological excavation is not decreased, but it is even increased in size. In these cases, the predominance of the inflammatory action being at the periphery of the nerve, a wall-like swelling surrounds the circumference of the disk. As this swelling extends into the retina, it raises the peripherical border of the nerve at the expense of the centre, and thus increases the depth and size of the excavation, especially if this is sufficient to put the central vessels upon the stretch. Such a condition as this is apt to occur in the graver cases of syphilitic neuritis, in which, on section, the sheaths are sometimes found enormously thickened through inflammation.

Here I would pause to say, what perhaps had been better said at the outset, and that is that neuritis, like retinitis, has no definite symptoms for definite diseases. It has certain elements of congestion of the vessels, of hyperplasia of the tissue, of hæmorrhage, and of œdema, and other signs and symbols of inflammation. Thus, the neuritis in syphilis may vary from the slightest shade of congestion or the merest trace of infiltration to the most flagrant form of inflammation, such as is seen in choked disk, and it may readily happen that the most expert examiner might not be able to detect any difference between the two, or to establish a diagnosis; and, when this is done, it is often not so much from the aspect of the nerve itself as from the behavior of its surroundings—the retina and the retinal vessels and the accompanying affections of the muscles—very much upon the principle of "Tell me who you go with and I will tell you what you are." I would also add that, after all, one glance at nature is worth more than all the pages of mere description that could be written. It is useful, however, as well as interesting, to know the factors which go to make up the picture, even if the multitudinous variations in it can not be adequately described.

The swelling which the nerve undergoes, which sometimes produces such a degree of protrusion of the head of the nerve, is caused by the varicose swelling of the nerve-fibres, the dilatation of the vessels, the hyperplasia of the connective tissue, and the œdema, as is very clearly shown in Fig. 47. The changes in the retina are those which occur in the graver forms of retinitis in which all the layers are

implicated. These changes have been described under retinitis, and it will be only necessary to remind the reader that the white plaques here, as there, are due to fusiform hypertrophy of the nerve-fibres, fatty degeneration of the tissue and capillary vessels, and serous and fibrinous exudations.

Fig. 47.

Retrogressive Changes.—The changes which take place in papillitis are those which would be expected in the subsidence of any inflammation. The œdema is gradually absorbed. The swelling abates both in the nerve and retina. The hæmorrhages become absorbed, and, when they are of large extent, their surface is broken up by small patches, of a yellowish-white color, some of which seem to be due to de-hæmatized blood, and others, from their brilliancy, to fatty degeneration of the fibres, in which may be included also the radial fibres of Müller. These yellowish patches may be interspersed with brownish patches or striæ, and thus produce precisely the same effect that the neuro-retinitis of Bright's disease does in the acute stage. These patches may be entirely absorbed or remain for a very long time, if not forever, especially when they are arranged in a circular or zonular form round the macula lutea, even increasing the resemblance to retinitis albuminurica. During the subsidence of the œdema and swelling, the vessels begin to come again into view, and to resume somewhat their natural form and aspect—first the veins and then the arteries—and one is surprised, often astonished, to see the degree to which they regain their normal condition and color. Indeed, this may be the case with the entire fundus. I have seen cases which seemed beyond redemption regain, after a longer or shorter period, an almost complete recovery, with little or no deviation from the normal standard, either as to the ophthalmoscopic appearances or the acuity of vision.

Of course, this is not usually the case, for commonly the optic nerve, as well as the entire fundus, has a worn or even battered air, which plainly shows the results of the struggle which it has been through.

The edges of the nerve are irregular and uneven, while those of the choriodal opening look frayed and discolored, with small collections here and there of pigment. The surface of the disk appears untransparent and dense, with a more or less dirty and discolored aspect. The capillary circulation is concealed to a great degree or entirely wanting. It is important to note carefully the signs round the borders of the disk, as they often form important diagnostic marks between the atrophy from papillitis, due to tumor of the brain, and the clean-cut and sharply defined border of the nerve in primary atrophy, or in secondary atrophy consecutive to a retro-bulbar neuritis.

Later on these changes are intensified: the vessels, moderately large in the early stages of resolution, become narrower, especially the arteries, and vision, which may have risen to a fair amount, now begins to fail, showing the influence of the contracting tissue upon the nerve-fibres. White streaks begin to appear along the vessels, and a veil-like covering of connective tissue may mask the porus opticus. This process of narrowing of the vessels may go on until they are of thread-like proportions, or entirely invisible. Sometimes, however, the veins in the retrogressive stage, instead of becoming smaller, may increase in size, and become even more tortuous than in the acute stage, which is probably due to elongation from atony of the walls.

The nerve now gradually passes into atrophy, which can not always be distinguished from ordinary atrophy, but which, nevertheless, as a rule, differs from it in some important respects, inasmuch as there is more tissue in the head of the nerve and a less tendency toward excavation. The white color is, too, much less brilliant, being often of a dirty-grayish tone, with little or no luster to it. The lamina cribrosa is veiled from view. There are also apt to be more changes in the retinal tissue, with the stellate figure at the macula. This is usually not so complete in form as in Bright's disease, so that it represents a segment rather than the whole or even the half of a circle. The individual spots are also smaller in size.

The vessels upon the nerve and in the retina are apt to be narrower and much more tortuous than in simple atrophy.

In descending neuritis, especially that from meningitis, the atrophy which follows is marked by the peculiarity that the disk is sharply limited, with but little disturbance at the choroidal edge, and shows less signs of previous inflammation than that which follows tumors of the brain. The vessels carry their normal calibre to a greater distance, and in some cases the lamina cribrosa can be clearly seen, while after choked disk, from tumor, the head of the nerve is fuller than usual, and the lamina is concealed.

Microscopical Changes. — In the first or congestive stages of neuritis there are no microscopical signs of inflammation. In true interstitial neuritis and peri-neuritis there is cell infiltration, with hypertrophy of the connective-tissue stage-work, especially that which carries the vessels, and especially, too, in the neighborhood of the central canal. The bulk of the nerve is increased by this and by infiltration of lymph-corpuscles, while the inclosed nerve-fibres either show no change whatever, or are at the most somewhat more richly provided with nuclei. Later on, however, atrophy of the nerve-bundles takes place, so that through pressure they become very much smaller, and finally, in the more serious forms, fade entirely away, so that at length only a connective-tissue string remains in the place of the nerve.

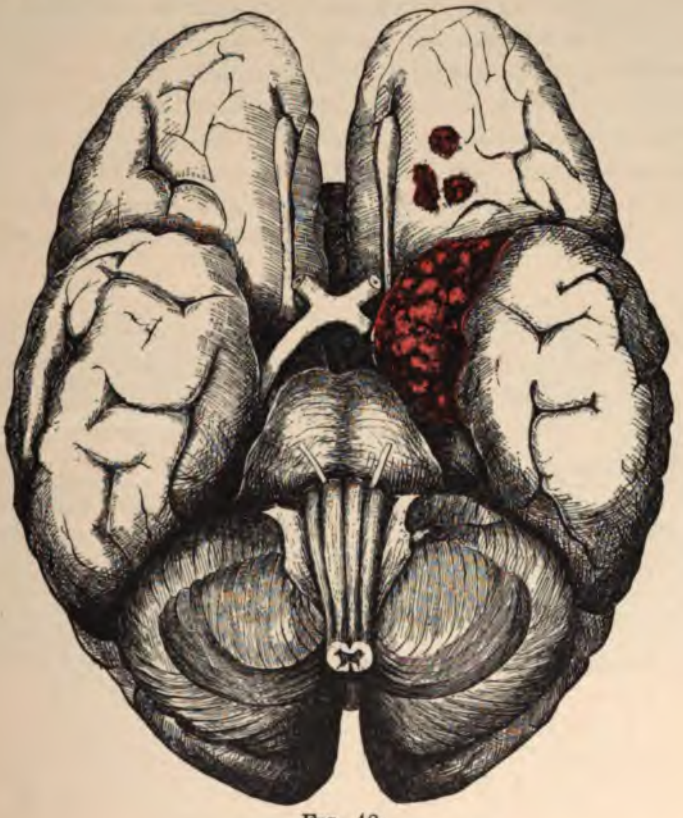

FIG. 48.

In other cases, with less thickening of the connective-tissue staging, gray atrophy ensues, in which the nerve-bundles are preserved as

such, but become smaller through loss of their medullary sheath. They assume a gelatinous appearance, which accounts for or rather occasions the discolorations seen with the ophthalmoscope in some forms of atrophy. This same gray degeneration is seen in medullary neuritis, with the presence of numerous fat-cells. Gray degeneration may also occur as an independent process and without any apparent hyperæmia. Indeed, it has been doubted whether there could be any true inflammation of the medullary substance, and whether it was not always rather a process of degeneration than inflammation.

In some forms of neuritis from brain-disease—that is, descending neuritis, which is accompanied by pronounced peri-neuritis and interstitial neuritis—the inflammation may be looked upon as a propagation of that of the connective-tissue elements to the medullary substance; and here the neuritic degeneration does not always show itself at the disk, but proceeds frequently from the chiasma down along the stem of the nerve or backward along the tracts.

The microscopical changes in choked disk are illustrated by the following figures, which were drawn from nature by Dr. H. C. Eno, in the case of a man who died from a sarcoma of the brain. Fig. 48 represents the base of the brain, the site of the tumor being shown by the red shading. Fig. 49 is a longitudinal section through the

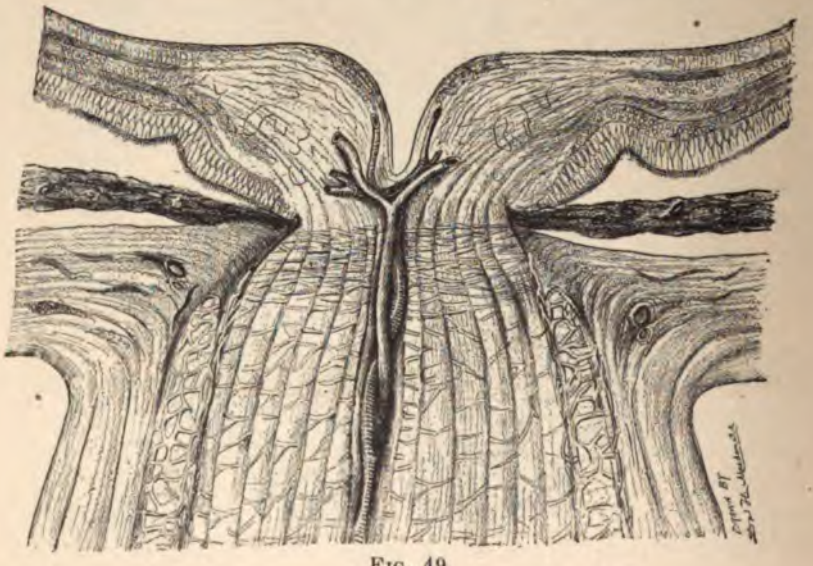

FIG. 49.

optic nerve and papilla. It shows the large degree of swelling of the optic nerve due to the varicose or ganglionic enlargement of the non-

medullated fibres, hyperplasia of connective tissue, œdema, serous congestion, and development of new-formed vessels and capillaries. These ganglionic swellings are sometimes of an enormous size and offer a homogeneous appearance of a yellowish color and contain many fatty and glittering granules, such as are found in various forms of retinitis. They cause the white lustrous effect which the nerve sometimes possesses.

Collections of lymphoid cells are also found. Fig. 49 also shows the distention of the sheaths. It, moreover, shows very clearly how

FIG. 50.

the layer of rods and cones is displaced outward by the swollen head of the nerve, and explains the fact pointed out by Knapp, that in cases of choked disk the blind spot is enlarged, sometimes enormously so.

It shows too, in this case, as in many others examined by different observers, that notwithstanding the large amount of swelling there was no choking of the disk or constriction of the vessels at the scleral

opening or elsewhere. Hence, although such constriction may take place, it is not essential to the process, and the name choked disk is not therefore applicable. Still, there are cases where the signs of constriction are evident enough. It must be remembered, too, that the mere fact that the signs of strangulation are not found after death is not enough to prove that the vein was not more or less compressed during life, or even if not compressed so as to alter its calibre to any degree, why there should not be a retardation of the current, which would produce a form of stasis with processes of irritation. Fig. 50 shows the ganglionic dilatation of the nerve-fibers.

Fig. 51.

Fig. 51 shows the swelling of the intraocular end of the optic nerve of the left eye, from a sketch made at the autopsy by Dr. Edward Curtis. It was formerly supposed that this distention of the sheath was due to an accumulation of fluid which came from the brain. It is more probably due to the fact that the neuritis is extended to this part of the sheaths and becomes a peri-neuritis. This is more probably the case, as this part is the most vascular portion of the nerve, containing, as it does, not only the vessels proper of the sheaths, but also ramifications from the central artery and branches from the short ciliary vessels through the scleral ring. This would also explain why, when the inflammation has gone so far as to affect the parenchyma of the sheaths in this part, there should be the cavernous openings in the lamellæ noticed in dissections. Fig. 52, which is from Schwalbe, shows the separation of the dural sheath into its four lamellæ, and their continuations into the sclera.

Fig. 52.

Fig. 53.

Fig. 53, also by Dr. Curtis, is a section of the posterior half of the eyeball, showing swelling of the nerve and papilla, and dilatation of the intra-vaginal sheath. Fig. 54 is an illustration by Dr. Eno of the ophthalmoscopic appear-

ances of this case of typical choked disk from tumor. The elevation, as measured by the upright method, being 1.09 millimetres as the refraction at the papilla was + 4 D. (see Vol. I., page 113).

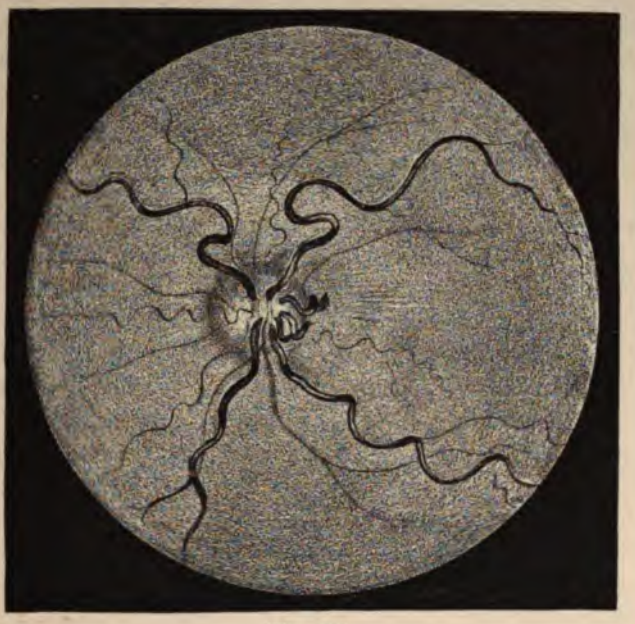

FIG. 54.

This swelling of the head of the nerve may attain, as has already been stated, a height of two millimetres above the plane of the choroid, and is usually accompanied by a hypertrophy of the outer layers of the retina, which also increases the elevation near the disk. Œdematous spaces may form in the outer layers as well as in the nerve-fibre layer, and if the papilla is much elevated it may carry the retina with it and thus cause a small separation of the retina near the head of the nerve. The white plaques in papillo-retinitis are like those in retinitis albuminurica, due principally to a considerable infiltration of granular cells in the granular and inter-granular layers, to which may also be added hæmorrhages, pigment-cells, and fatty degeneration.

The walls of the vessels are thickened and the adventitia is hypertrophied, and the media in the later stages much increased in thickness, due to sclerosis.

In the atrophic stage the nervous elements disappear still more, and the papilla becomes flatter. Still, it may even in the atrophic stage project above the retina, especially when there has been much

hyperplasia of the connective tissue, as the retina itself contracts below its usual level.

The changes in choked disk, so far as the microscope has yet shown, may be limited by the lamina cribrosa, and may be fairly called intra-ocular. They may, on the other hand, extend to the sheaths and advance up the stem of the nerve, becoming a neuritis ascendens. Or, on the contrary, they may progress downward from the brain along the nerve and sheaths, and form a neuritis descendens.

Visual Disturbances in Choked Disk.—The amount of vision in choked disk may vary from a normal acuteness to total blindness. Progressive amblyopia commonly occurs as the disease continues. The central vision is affected, as well as the peripherical. The field usually contracts most upon the nasal side, until the whole inner half of the field has gone, as well as the centre, and all that remains of it is a small eccentric part outward. Certain defined limitations of the field may occur, of course, from direct pressure of a growth upon the nerve, that may then resemble hemianopsia. Marked and sudden blindness, with dilatation of the pupil, when it occurs with choked disk, is not due to neuritis alone, but to direct pressure upon the nerves by a growth, or from distention of the third ventricle.

Differential Diagnosis.—The diagnosis between the various forms of neuritis can not always be made with the mirror. Still, there are some differences which, if not always present, are so to a predominating degree, and which sometimes serve to draw distinctions between the varieties named above. Thus, in the ascending form and that which usually accompanies choked disk, from tumors of any description, there is usually a much more pronounced degree of swelling and inflammatory action than in the descending form. Moreover, in papillitis the protrusion of the disk, besides being more prominent, is limited more to the head of the nerve itself, presenting a steep and abrupt descent to the retina, thus giving to the papilla the mushroom appearance. The greater this peculiarity is, the greater the probability that the process is due to tumor or growth of some sort in the brain. On the other hand, a more moderate degree of swelling, and a more gentle and further reaching implication of the retina, are due rather to a neuritis descendens from meningitis than tumor—unless, indeed, the meningitis is of the chronic form, when the symptoms resemble or are identical with those from tumor. Again, the white plaques are apt to be smaller and more glittering in neuritis descendens than from tumor.

Thus, I have not infrequently seen, in meningeal troubles, a low form of neuritis with very little inflammatory action, and which, on

further progress of the disease, turned into a marked form of neuro-retinitis, in which the swelling of the nerve passed into the retina, with no marked difference in level, and with a tendency to extend itself rapidly toward the periphery. In basilar meningitis there is at times a glittering effect, that extends over the disk and into the retina, especially along and over the vessels, which is not present in other forms of neuro-retinitis. Still, it must be borne in mind that these peculiarities are not always present in neuro-retinitis from meningitis, and that there may be a genuine form of choked disk of the most pronounced character.

As a rule, too, vision is usually much more seriously affected, and affected earlier in neuritis descendens than in ordinary choked disk. It is apt to be affected, too, before marked ophthalmoscopic changes have occurred at the disk. Indeed, these may not appear for months, or even years, and then only in the form of a secondary atrophy.

The pupil, too, is apt to be very sluggish or unevenly dilated on the two sides, or abnormally dilated on both sides, while in choked disk the pupil and vision are much less likely to be affected in the early stages of the trouble; indeed, may not be so, even when the inflammation is at its height, and the ophthalmoscopic signs have been fully developed for a long time. Thus many cases with marked or even exaggerated ophthalmoscopic signs have been reported, which have been watched for years, in which the vision remained normal. Mauthner reports a case which was under his and Becker's observation for three years; and Dr. Rushmore reported a case to the New York Ophthalmological Society that he had had under observation for ten years. In Mauthner's case, the microscopic examination, made by Iwanoff, showed that the swelling was due to an increase of vessels, serous infiltration, with moderate hypertrophy, but with little or no increased cellular infiltration. The nerve-fibres themselves were in a normal condition. Moreover—and this is most important—the process was limited by the lamina cribrosa, beyond which it did not extend.

The differential diagnosis in the papillitis, dependent upon tumor of the brain, and that due to Bright's disease, has already been given under retinitis albuminurica (Vol. II., p. 149).

Neuritis due to tumor in the orbit is usually differentiated from that occasioned by an intracranial growth, by the fact that, besides being confined to one side, there is usually some exophthalmus. Double vision may, of course, be present in either case.

It is not within the scope of this book, which deals with the features of disease as they exist, rather than with the causes which

led to their production, to dwell at all upon subjects which are still matters of speculation. Still, it may be of service to the student to refer in a very brief manner to some of the more prominent theories which have been advanced in the explanation of such important pathological processes.

Causes of Papillitis (Choked Disk).—The first important theory in the explanation of choked disk was that offered by Von Graefe. This implied an increased intra-cranial pressure, which extended its influence to the cavernous sinus. The result of such pressure would be an impediment to the return circulation from the retinal veins through the ophthalmic vein to the sinus. A backing up of the current would thus occur, especially at the constriction formed around the head of the nerve at the scleral opening (see Vol. I., p. 32). This stasis would lead to irritation of the surrounding parts, until almost complete strangulation took place in this part of the nerve, which would be followed by all the peculiar ophthalmoscopic signs and morbid appearances shown in microscopic sections *

Benedikt's Theory.—This is that some irritation is brought to bear on the sympathetic nerves, and that this irritation is propagated in some subtle way to the vaso-motor filaments which regulate the circulation of the disk—in other words, that the neuritis is one of abnormal innervation of the sympathetic system.†

Schmidt's Theory.—This assumes that choked disk is the result of the intracranial fluid being forced down between the two sheaths of the nerve, which, when it arrived at the ocular end, occasioned stasis of the circulation and all the phenomena expressed in papillitis.‡

Parinaud's Theory.—This was a modification of Schmidt's, inasmuch as it assumes that it was not necessary for the fluid to be forced down or to migrate from the subarachnoidal space of the head into the corresponding space of the optic nerve, but simply that a stoppage in its flow should take place from intracranial œdema, caused by distention of the ventricles of the brain. This arrest of the flow of the lymphoidal fluid through the lymph spaces of the optic nerve produces a stasis of the blood-vessels and all the attending manifestations of choked disk and distention of the sheaths. §

Hughlings Jackson would seem to favor substantially Benedikt's theory, since he thinks that optic neuritis may be "a doubly indirect result of local gross organic disease; that first there are changes of

* "Archiv für Ophth.," B. XII., ab. ii., p. 100.
† "Elektrotherapie," 1868, p. 249; ibid., 1876, p. 404.
‡ "Archiv für Ophth.," B. XV., ab. ii.
§ "Annales d'Oculistique," p. 82, 1879.

instability about the tumor, that next these lead on discharges, by intermediation of vaso-motor nerves to repeated contractions with subsequent paralyses of the vessels of the optic nerve centres, and at length to that trouble of nutrition which is optic neuritis." *

According to Gowers, Galezowski believes that neuritis is always descending, and Galezowski first maintained, contrary to previous observers, that the intra-ocular change is in all cases the visible manifestation of an inflammation propagated by continuity from the brain. Gowers, and some of the more modern observers, would seem to favor this view. It is, however, directly opposed to the microscopic results of such observers as Von Ammon, Virchow, Schweigger, and Iwanoff. The question is not whether the stem of the nerve is found diseased in connection with choked disk, but whether choked disk can exist without it, and the authorities mentioned above state directly that it can, and that the microscopical changes may discontinue at the lamina cribrosa, the rest of the nerve being entirely normal.

In 1882 the writer advanced the view that "choked disk, in connection with brain-disease, is not simply a vaso-motor disturbance which leads to inflammation and stasis on account of the anatomical condition of the parts affected, but that it is a true neuritis, which is the expression of an irritation of certain fibres of the fifth pair—fibres which not only regulate the blood-supply in the vessels, but which also maintain the healthy processes of waste and repair of the elements of the tissues themselves. Furthermore, that the irritation is conveyed, not by the isolated fibres of the sympathetic system, but through the agency of the trigeminus, and especially of its recurrent ophthalmic branch." †

It will be seen from all this that the true cause or causes which lead to choked disk are not yet definitely established.

Retro-bulbar Neuritis.—Among the milder forms of neuritis is one which, from the doubt as to its true nature and from the frequency with which it occurs, is one of the most curious as well as most interesting forms of inflammation of the optic nerve. This affection would seem to vary between a neuritis or neuro-retinitis and atrophy, or to partake of all three characteristics at the same time. It has, for this reason, been designated by Leber as a form of chronic retro-bulbar neuritis with partial atrophy of the optic nerve, "in which the atrophy shows itself in the outer or temporal circumference of the disk."

* "Times and Gazette," March 19, 1881, p. 316.

† "New York Medical Journal," June, 1882.

The changes seen by the mirror are usually a moderate degree of injection of the smaller vessels of the nerve, which sometimes scarcely passes the physiological limit, and then again is more marked. In this case, it shows itself in such a way that the middle zone of the nerve is more congested than the other portions. The congestion then extends to the central portions. In this way, the connective-tissue ring, instead of being more or less concealed, is rendered more conspicuous.

With this injection of the optic nerve there may be a very delicate and hardly perceptible haze or indistinctness of the tissue of the nerve, which may at times extend itself into the retina. This slightly veils the contours of the nerve, resembling only to a less degree what is seen in diffuse retinitis.

The larger vessels are but little, if at all affected, either in their size or course. Occasionally, however, the arteries are reduced in size, especially near the disk. As a great rarity, the veins may be slightly dilated and somewhat more tortuous than usual; but what is much more constant, and, at the same time, much more important, is the reduction or the total absence of the light-streak on both arteries and veins, showing that the larger vessels are, to a degree at least, implicated in the process, which is also made evident at times by the fine white lines extending along the borders of the vessels. Leber is, indeed, of the opinion that some of the cases of perivasculitis belong to this category of diseases. He further believes that it is not improbable that similar changes may take place in the vessels of the stem of the nerve, as had been, indeed, already suggested by Mooren.

If this is so, it may be, after all, that the initial lesion may lie with the vascular apparatus, and that the subsequent atrophy of the fibres is due to an altered blood-supply in those vessels which nourish the particular fibres that are distributed to this part of the retina, which is marked out by the central scotoma. Anatomy teaches us, moreover, that the axial fibres of the nerve pass from the central portion in the stem to the outer part of the disk on their way to be distributed to the retina. This being so, it might have an important bearing on those forms of general nervous disease—notably spinal affections, where low and chronic forms of optic neuritis are so common; where, too, in the author's opinion, contrary to the general belief, the atrophic stage is usually, if not always, preceded by a congestive stage.

Besides these signs of congestion, there is that phantom-like haze over the entire fundus which has been so often spoken of in these

pages, and about the true seat of which it is impossible to say whether it is in the nerve-layer or in the middle layers, where the capillary network is most pronounced, or whether it is due to the participation of the choroid.

Accompanying these signs, and even in the congestive stage, and before there are any visible signs of atrophy, there may be, and usually is, a peculiar scotoma, which extends horrizontally from the point of fixation to the nerve, including the blind spot. With this peculiar scotoma there may be marked central amblyopia with a normal excentric field, or, in very rare cases, the central vision may be good and the periphery affected. Even when this central scotoma is not well enough defined to be an actual defect, there is almost universally a color scotoma, especially for red, and this may exist even where there is very little central amblyopia.

Following the congestive stage, which may be so slight as to be hardly perceptible, comes a moderate degree of atrophy of the outer circumference of the nerve. This de-coloration is, as a rule, limited to the outer half of the nerve, and only in very rare cases and those of a very pronounced and inflammatory character, does the blanching of the nerve extend over to the nasal side. There may be, too, a slight atrophic excavation of the temporal side of the nerve. As a result of this atrophic condition, the meshes of the lamina cribrosa are sometimes brought to view or rendered more conspicuous.

Leber first suggested that this form of the scotoma was due to the implication of those fibres, which he claimed have a peripheric position at the disk, and which are then distributed to the section of the retina which lies between the papilla and the macula, and which are finally arranged in such a way as to correspond in shape and position to the scotoma. Later investigations by Förster, Nettleship, and Edmunds would seem to corroborate Leber's view. They, however, go further, and show that those fibres which are situated at the circumference of the nerve when they have reached the disk have a central position in the more posterior parts of the nerve—that is to say, that what are "axial" fibres in the posterior parts of the nerve become peripheric fibres at the disk. Thus, a central scotoma may be the result of an axial neuritis if the inflammation is in the posterior parts of the nerve, and of a peripheric neuritis if situated anteriorly. Leber believes that his explanation of the central scotoma is applicable to all forms of neuritis where a scotoma of this character exists.

Perineuritis and Hydrops of the Sheath.—Strictly speaking, perineuritis in the sense of an inflammation of the surrounding

sheaths of the optic nerve, can not be seen with the ophthalmoscope, until by propagation it has become an intra-ocular neuritis. As such it increases the swelling of the papilla, and aids oftentimes in forming a sort of wall around the circumference of the disk, which has already been described in connection with retinitis and optic neuritis, forming what is known as retinitis circum-papillaris. The same is true in regard to the non-visibility of the appearances, so far as the ophthalmoscope is concerned. A cone-like distention of the sheaths of the nerve occurs very frequently with neuritis, especially with that kind which is known as choked disk; but hydrops of the sheath may exist as an independent disease, which consists of a collection of fluid between the two sheaths. This fluid may, after a shorter or longer time, usually the latter, produce a neuritis through irritation. This then shows itself at the disk like the ordinary neuritis, but is usually of the more marked forms of papillitis, such as choked disk, from tumor of the brain.

Hæmorrhage into the intervaginal space produces at times the same effect (see Vol. II., page 97). Manz found hydrops of the sheath frequently in meningitis without the complication of neuritis, which shows that it requires time for the development of the papillitis, as it does also for descending neuritis and peri-neuritis.

It will readily be understood that a retro-bulbar neuritis may exist in any part of the nerve behind the eye. When this is the case, it is beyond the reach of the mirror, and only gives evidence of its existence by the effect upon the vision.

It often happens, however, that some time after the deterioration of sight, signs of neuritis begin to make their appearance at the disk, and the affection is changed then by continuity or descent from a retro-bulbar into an intra-ocular or ordinary neuritis, with at times a marked redness and striation, but with little swelling, œdema, or hæmorrhage.

The neuritis usually occurs on one side, or, if in both eyes, the second is commonly affected after a lapse of time, which may be expressed by an interval of weeks, months, or even years. Instead, however, of the signs of a neuritis which has advanced along the sheaths and substance of the nerve, the first ophthalmoscopic appearances visible may be those of secondary atrophy, the result of the inflammation in the more posterior portions of the nerve.

Such being a short description of some of the forms of neuritis as they most commonly occur, it remains to give a glance at some of the conditions under which the appearances are produced.

In the first place, it is essential to remember that neuritis may oc-

cur as an idiopathic affection, and that as such the widely different features it presents at different times, are not distinctive forms of different processes, but different phases of the same process, and that while very dissimilar in their aspects they are all of the same nature and resemble each other very much as the mist resembles the rain, or, to carry out the metaphor, as the breeze does the storm.

The milder forms of neuritis are usually met with as the result of over-use, exposure to glare, or are associated with any constitutional disorder, such as rheumatism, gout, syphilis, menstrual disorders, typhus or typhoid fever, and other febrile disturbance. When, however, the neuritis or neuro-retinitis occurs in cases of fever, especially where there is delirium, there is reason to believe that there is meningitis combined with the fever. Von Graefe was inclined to consider some of these cases, that occurred after fever and other troubles, and in which the principal if not the sole ophthalmoscopic sign was a marked narrowing of the arteries, as due not to embolism of the central artery, but to a retro-bulbar neuritis, with compression or thrombosis of the vessels. The fact, however, that in such cases the trouble is usually double-sided is against either embolism, retro-bulbar neuritis, or thrombosis, especially as a complete restoration of vision often occurs as suddenly as it was lost. Such cases are probably due, principally at least, to ischæmia of the retinal vessels, as has already been described. The same form of blindness sometimes occurs after pregnancy, pneumonia, and other serious diseases, and with the same obscurity as to its cause.

Occasionally we also see these milder forms of neuritis, either as the accompaniment or sequelæ of scarlet fever, measles, and other exanthemata, but often here we get the graver forms, such as choked disk with implication of the retina, which is oftentimes, no doubt, dependent upon renal disturbance, even if no signs of it are present in the urine, or upon some cerebral conditions, especially meningitis.*

Among the milder, if not among the mildest, forms of neuritis are certainly to be classed those which come from the abuse of alcohol and tobacco. Indeed, the symptoms are often of so trivial a character as to have led to the assumption that the amblyopia often exists without any visible ophthalmoscopic signs. Still, I am more and more of the opinion that, to a careful observer, there are always, or nearly always, some perceptible even if indistinct changes apparent, as has already been stated under the description of retro-bulbar neuritis. In this category must also be placed the effects of other toxic

* For some interesting cases of neuritis after measles, see a paper by Wadsworth, "Trans. Amer. Ophth. Soc.," 1880, p. 125.

agents, such as lead; but it must be remembered that these diseases may at times be the cause of the graver forms of neuritis or neuro-retinitis.

Neuritis from Hereditary and Congenital Influences.—This affection must surely be classed among the milder forms of neuritis. Indeed, so little marked are the primary symptoms of congestion that oftentimes it is not until the secondary or atrophic stage has commenced that attention is called to the trouble by failure of vision, and the disease has consequently been spoken of by some authors as primary atrophy of the optic nerve.* Nevertheless, I have seen cases, and one in particular, in the very first stages of the disease, where there were marked signs of congestion of the disk, with conspicuous striation of the papilla and surrounding retina.

The case just alluded to was that of a young man of twenty years, and who was one of a family in which the trouble had been known to exist for three generations and in which crossed heredity showed itself, the male members being attacked in one generation and the female in the next. According to Leber, the disease almost always is confined to males, which is no doubt true in general; but, besides the above instance where the female members of the family were affected, I saw, through the kindness of Dr. R. H. Derby, a perfect specimen of the disease in two sisters of middle life, in whom the trouble appeared almost at the same moment. In these cases there was the same central scotoma and the same mild form of neuritis which has been so often spoken of as occurring with retro-bulbar neuritis.

It seems singular at first sight that the particular fibres which preside over central vision should be those that are attacked in all that large class of cases, which are supposed to be due to retro-bulbar neuritis with partial atrophy. This may be dependent upon the fact that, as the fibres are the most sensitive in function, so they may be the most delicate in organization, and therefore most susceptible to morbific influences and the first to suffer, and this would appear to be all the more likely, as later on in the disease or in the more pronounced degrees a central loss of vision is not uncommonly accompanied by a concentric narrowing of the field. Even if this was not true, the predilection of certain fibres to certain influences of a morbid nature is not so rare in nervous pathology as to excite surprise. It is also curious enough to deserve attention, that the presence of sugar in the urine, as, for example, in diabetes, should produce the same central

* "Hereditary Atrophy of Optic Nerve," W. T. Norris, "Trans. Amer. Ophth. Soc.," 1885, p. 662.

scotoma and the same indefinite ophthalmoscopic signs as alcohol and tobacco, so that it will be seen that there is a large class of diseases of very diverse natures that are followed by the same set of appearances which are symptomatic, not of the diseases themselves, but of the locality affected.

Neuritis in Uterine Disorders.—This is usually of a mild form, when not associated with renal trouble which produces retinitis albuminurica. The ophthalmoscopic appearances commonly show a mild injection of the optic nerve in the early stages, with sometimes a trace of diffuse retinitis near the disk. These appearances are not at all in proportion to the disturbance in vision which may at times show a central scotoma, as in retro-bulbar neuritis; or the amblyopia may extend over so much of the field and be of so pronounced a degree that only a small amount of eccentric vision remains.

As in other forms of what is called retro-bulbar neuritis, the first symptoms presented to the physician may be those of atrophy. The following is a typical case of this kind:

A young woman of twenty-one had, some two years before examination, sudden suppression of the menses. This was followed by a rapid loss of sight in the left eye. She had been regularly treated for this in an ophthalmic hospital without improvement. A week before visiting me the same thing happened again, the right eye being attacked this time. Examination showed the vision in this eye to be reduced to one sixteenth of the normal quantity, $V = \frac{1}{16}$; that in the left eye was reduced to counting fingers in the outer part of the field. The left eye, which had been attacked two years before, showed fully-developed atrophy. The right showed signs of a mild neuritis, with increased capillary injection of the centre of the nerve. There was a central scotoma of a light degree, the rest of the field being intact.

Under vigorous and systematic leeching and other derivative measures, followed by large doses of iron, the menses were reinstated in a short time, and vision gradually improved until it rose to the normal ($V = 1$).

The patient then married. She had a good deal of trouble with her first confinement, and a few weeks after, while nursing her infant, vision again began to fail.

The child was at once weaned, and everything done to bring on the menses. When these appeared, vision began to improve, and at the end of nearly a year was again normal in that eye, the left remaining permanently blind. The ophthalmoscope showed complete atrophy in the left eye, with some blanching of the nerve of the right

eye. This was combined with marked signs of what is known as retinitis albescens.

It is hard to imagine to what such cases are due, if not to some form of neuritis which is occasioned by a reduction in vital force, and which shows its effects in the peripheric parts of the nerve, just as it sometimes does in other nerves. I am inclined, therefore, to think that what is so often spoken of as uræmic blindness without ophthalmoscopic appearances is only this form of neuritis, especially in pregnant women, and in which the organic change is so slight as to escape notice, but which, nevertheless, is sufficient to interfere with the proper function of the nerve or to annul it entirely.

These cases of uterine amblyopia are so important, and practice is so much better than precept, that I am induced to cite another case, especially as I believe that the priceless boon of sight might be preserved to many women by vigorous measures, who have been and will be doomed to blindness.

E—— B——, thirty-five years old, consulted me for failing sight. She had been married eight years and had had three confinements. Her attention had been attracted to her eyes about two weeks before her first confinement, when she noticed an imperfection of sight upon the "left side of her left eye," which seemed like a dark band upon a light ground. After her confinement the bright ground disappeared, but not, however, until her menses recommenced. The dark band, however, remained, and no object could be seen when carried beyond a certain distance to the left.

Just before her second confinement the same thing occurred, and the dark band increased in breadth so much that central vision in this eye was impaired to such a degree that it was of little use to her. Up to this time the right eye had remained intact. Just before her third confinement, however, precisely the same thing occurred in her right eye. She came to consult me three months after her confinement, while her vision, as in the other cases, began to fail two weeks prior to it. At the time of the examination the vision in her right eye, the only one remaining, was so reduced that she could barely count fingers at a few feet. The optic nerve gave the picture of white atrophy of that peculiar kind in which the capillary circulation seems to have suffered the most. In neither eye were there any capillaries to be seen upon the disk, while the larger vessels seemed to have preserved their normal relation as to size and course. No trace of any kidney trouble was found, the urine was free from albumen, and the general health was perfect.

There was, notwithstanding all medication, no change in the vision

until at the end of a month, when the courses returned, and from that moment a gradual improvement took place, until vision rose to one third of the normal quantity $V = \frac{1}{3}$. The patient was dismissed, with an emphatic warning not to become again pregnant.

Eight months later, however, this took place, and as there was an almost absolute certainty of blindness ensuing if gestation was allowed to pursue its course, premature delivery was produced. This was the first time, so far as I am aware, that premature delivery was ever performed for the sole object of preserving the sight, which happily in this case, as it has been in so many subsequent ones, was successfully accomplished, as two years subsequent to this the vision had not deteriorated but improved.*

Simple neuritis may occur at the climacteric period; but at this time the disturbance usually shows itself as neuro-retinitis or neuritis-hæmorrhagica, which is sometimes accompanied with marked cerebral disturbances and much swelling of the disk. It should also be borne in mind that what takes place with irregularities of the menstrual flow may also occur in the disturbances of other secretions, such as suppression of the urine or sudden checking, either spontaneous or induced, of long standing and chronic discharges.

Rheumatic Neuritis.—This is usually a light papillo-retinitis, with pain upon pressing the eyeball back into its socket, which is probably caused by a light periosititis. It is not usually accompanied by any paresis of the ocular muscles, as none of the nerves pass through the optic foramen. In these cases and many others like them, where the arteries are very much reduced in size, the diagnosis lies between retro-bulbar neuritis with compression of the arteries, and simple ischæmia of the retina.

Syphilitic Neuritis.—Inflammation of the optic nerve occurs very frequently with syphilitic affections, and may assume any form, from the mildest to the most grave.

In the milder forms it usually appears associated with or passes into a low degree of diffuse neuro-retinitis. It is apt to affect the sheaths of the nerve and cause peri-neuritis, combined with intraocular neuritis. This produces oftentimes a marked picture of papillitis (choked disk), with circum-papillary retinitis, due to hypertrophy of the outer layers of the retina in the neighborhood of the disk. In this case the ophthalmoscopic appearances are very similar to those due to tumor of the brain; and, indeed, it may well be that with the ocular affections there are also gummata of the brain, which are in

* "Premature Delivery for the Prevention of Blindness," "Trans. Amer. Ophth. Soc.," 1882, p. 423.

fact tumors and produce the same effect. Still, if there are no sensory disturbances and no paresis of the ocular or bodily muscles, the chances are that the neuritis is not due to a cerebral cause.

Neuritis from Cerebral Disorders.—Caries of the bones of the skull may be accompanied by neuritis, which then usually takes the form of choked disk like that due to tumor of the brain. Dr. Rushmore reported a case to the New York Ophthalmological Society where choked disk had existed for ten years from caries of the parietal bone.

Fractures at the base may produce either papillitis or white atrophy, as may blows and contusions. Thus, many years ago, I saw a case where a man had received a blow from a hammer upon the forehead and just above the right eye. There was extensive rupture of the choroid on this side. At the time of the first examination the left eye appeared intact and vision was normal. Two weeks later neuro-retinitis began to develop itself in the left eye, which passed into a violent form of choked disk, which, curiously enough, did not involve the right, in which the effect of the violence manifested itself in the form of rupture of the choroid. This would go to show that the inflammation or irritation produced by the blow was in some way, either through the bones or through inflammation of the meninges, extended to the other eye. There were, however, no cerebral symptoms whatever, or, indeed, any general disturbance. Unfortunately, the case was lost sight of after a short time.*

Neuritis from Meningitis.—This is not as frequent as is commonly supposed, and when present is usually of a milder form than that due to tumor of the brain; and, indeed, the inflammation may show itself as a mild form of neuro-retinitis, or be limited to hyperæmia or even simple irritation of the retina and nerve. When this is confined to the disk, the injection seems to lie on a deeper plane than the retina, while the connective-tissue ring is not only not obscured, but even made more conspicuous in contrast with the more central congestion.

Neuritis from meningitis is usually accompanied with marked cerebral disturbance, pain, and deterioration of vision, and by dilatation of the pupil. In acute meningitis, neuro-retinitis is more apt to occur than choked disk. In the chronic form a descending neuritis is apt to be produced which at times shows itself as simple neuritis, with marked striation of the nerve and more or less hæmorrhage near the disk. When, however, peri-neuritis near the globe has occurred,

* See also "Optic Neuritis in Head-Injuries," Edmunds and Lawton, "Trans. Ophth. Soc. of the United Kingdom," vol. vii., p. 208.

with hydrops of the sheath, choked disk is more common. Indeed, this latter may be present even in the chronic forms from the beginning, but the tendency is the other way.

With acute tubercular meningitis neuritis is more common than with the other forms, and here it usually assumes the form of choked disk. Sometimes neuritis may occur in epidemic cerebro-spinal meningitis, which is a deviation from the usual occurrence of purulent choroiditis with pan-ophthalmitis.

In some instances children may have either single or double neuro-retinitis without any general trouble that can be detected. According to Leber, the presence of the neuritis would lead to the supposition that there was a latent meningitis. It has occurred to me at times that, as these cases are often associated with what appears to be a perfect state of health, the neuritis might at times be due to some hereditary taint, perhaps syphilitic, especially as such cases are improved by anti-syphilitic remedies. I have, moreover, seen this form of neuro-retinitis in young girls about the commencement of the menstrual period, and I have been inclined to look upon the trouble as one analogous to the retinal hæmorrhage which is apt to occur in boys about the age of puberty.

In grown people there may be also chronic neuritis due to circumscribed basilar meningitis, as shown at the autopsy. Here, however, there are usually pain, fever, and sleeplessness. The mirror reveals then commonly a mild form of neuro-retinitis which passes later into atrophy; or this latter may be the first ophthalmoscopic sign, as the neuritis may have affected only the intra-cranial parts of the nerve, and only show itself later in the eye as a secondary atrophy.

Neuritis from Hydrocephalus.—According to Von Graefe, in ordinary hydrocephalus where there is distention of the cranium, from yielding of the bones to pressure, the ophthalmoscopic signs are not those of neuritis, but of atrophy of the nerve. Allbutt suggests, however, that perhaps Von Graefe was not in the way of seeing these cases in their early stages, or until the neuritis had passed off and atrophy set in with loss of vision. Allbutt also mentioned having not infrequently met neuritis of the choked-disk variety in young hydrocephalic children. In several cases of enormous distention which I have examined myself, where there was no complaint as to sight, the eyes have been normal. In internal hydrocephalus, where the ventricles are distended, we sometimes see choked disk, and sometimes merely white atrophy, from pressure upon the chiasm, with a widely dilated pupil and great loss of vision. The edges of the optic nerve

in these cases are not like those of atrophy from a preceding choked disk, but like those of primary atrophy, being clean cut and well defined, and do not show that frayed appearance of the choroid near the borders of the papilla, nor are the vessels, as a rule, accompanied by white lines or other signs of inflammation.

Neuritis from Tumor of the Brain.—The neuritis which results from tumors is almost universally of the graver types, and usually in the form of choked disk. The growth may be of any character, and may occupy any position within the cranium, from the anterior portion of the skull to the medulla oblongata, and may be of any size, from a hazel nut to an orange.

The ophthalmoscopic appearance which this form of neuritis offers, and the differential marks between it and other varieties, so far as they exist, have already been dwelt upon in the description of choked disk. Still, a gentle reminder to the student not to put too much reliance upon these distinctive points may not be out of place. Choked disk is choked disk, and may come from a multitude of causes.

Abscess of the Brain.—This does not often produce neuritis, still it may do so occasionally. Thus, I have seen a light form of neuritis with obscuration of the edges of the nerve, and slight infiltration of the retina, from an abscess in the posterior lobe of the brain, and cases of choked disk have been mentioned as occurring from this cause.

Softening of the Brain.—This rarely leads to neuritis, although this occasionally occurs. In a recent case seen by me at St. Luke's Hospital, where the autopsy showed extensive softening, there was for a considerable time during life a marked neuro-retinitis with choked disk. There was, however, a trace of albumen in the urine, and the ocular symptoms may have been due to this. In these mixed cases of cerebral and kidney trouble, which occur oftener, I think, than is commonly supposed, it would at times be impossible to say whether the ocular symptoms were due to the cerebral or renal affection.

Thrombosis of the Sinuses.—Dr. Knapp pointed out, many years ago, that this may be accompanied with choked disk, which may, however, be the result of the accompanying meningitis. Among the more distinctive marks of the trouble are chemosis of the conjunctiva and protrusion of the eyeball.

Neuritis from Spinal Disease.—Neuritis from spinal troubles is almost invariably of the milder forms—so mild, indeed, that many observers think that the only manifestations disclosed by the mirror in locomotor-ataxy are those of primary atrophy. I am of the opin-

ion, however, that very often, if not as a rule, there is a congestive stage, no matter how light, which signifies an inflammatory process, and which precedes the atrophic stage. Thus, I have frequently seen a perceptible congestion of the optic disk which was followed later by atrophy, or a palpable injection of one disk, while the other showed the signs of an already beginning atrophy.

This congestion is of that peculiar form where the centre, or the central zone of the disk becomes injected. The increased redness then seems to lie in a deeper-seated plane, while the connective-tissue ring is not concealed, as a rule, but is sometimes even more conspiçuous than normal. Allbutt has also mentioned this initial stage of congestion, while others have been inclined to doubt that it ever occurs. The same mild form of congestion of the disk occurs not infrequently, if not as a rule, in general paresis.

Neuritis from Orbital Disease.—Phlegmons of the orbit, such as cellulitis, periostitis, growths, exudations, and hæmorrhages, may lead, through propagation of continuity, from a retro-bulbar to an intra-ocular neuritis, which may vary in form from a marked narrowing of the central vessels with occasional pulsation of the vessels, but with little or no swelling of the tissue, to a fulminating neuritis which is expressed by choked disk with thrombosis of the central vessel or stasis hyperæmia. This occurs, as pointed out by Graefe many years ago, especially in erysipelas of the face. (See also "Perivasculitis," Vol. II., page 27.)

Neuritis due to tumor in the orbit is usually differentiated from that due to an inter-cranial growth by the fact that, besides being confined to one side, there is usually some exophthalmus, with perhaps some limitation of the motion of the eye, which differs from that due to paresis of the muscles. Double vision may, of course, be present in either case. The diagnosis of an orbital affection is also corroborated somewhat when there is total loss of sight on one side and no reduction in the amount or change in the field in the other eye.

It is not within the scope of an elementary text-book upon ophthalmoscopy, which should deal principally, if not exclusively, with the manifestations of disease, to dilate much upon the causes which have led to them. The above sketch, like the proverbial straw, is given, therefore, solely with the view of helping the student, so far as it may, to form a possible if not a probable diagnosis. I would, therefore, refer the reader for information as to the medical side of the question to the admirable works of Allbutt and Gowers, and the almost countless monographs and papers which have from time to time appeared in the leading ophthalmoscopic journals.

CHAPTER VI.

ATROPHY OF THE OPTIC NERVE.

Atrophy of the optic nerve may occur in any of its parts from the intraocular end to its centres. It may extend from any given point peripherically toward the eye or centripetally toward the origin of the nerve. Atrophy of the optic nerve may come from many causes, but the ultimate result is the wasting away of the nervous elements.

Atrophy, as seen with the mirror at the disk, is recognized under various forms, the terms of which are somewhat puzzling, and apt to be used in a confusing manner.

Primary or simple atrophy, or simple primary atrophy, or, again, simple progressive atrophy, are terms used by most writers to express that form of atrophy in which the initial or primary wasting away of the nerve-substance has not been preceded by any visible signs of inflammatory action; or where, if such signs do exist, they are so trifling in proportion to the loss of vision as to be hardly recognizable, and still within what most observers would call physiological limits. Yet it must be borne in mind that the first symptom of atrophy is failing vision, which, from the fact that it usually attacks the periphery of the field, may have existed for a long time, and may therefore have been preceded by some inflammatory or congestive stage, which had come and passed long before. I am convinced in my own mind that the great majority of cases of so-called primary atrophy, if seen early enough, and examined with sufficient care, would show evidences of irritation and congestion of the nerve, even in those cases of spinal disease where it is sometimes asserted that no trace of inflammation is ever visible.

This condition of preliminary congestion has been admitted to occur in very exceptional cases, even by those who deny its existence as a rule, or even as an infrequent occurrence; and they have applied to it the rather inconsistent and contradictory term of congestive atrophy.

Secondary Atrophy.—If primary atrophy means a wasting away

which has not been preceded by inflammatory symptoms visible at the disk, it would certainly seem as if secondary atrophy ought to be that which had followed or was secondary to such inflammation. Secondary atrophy of the optic nerve, however, means an atrophy which is secondary to some lesion, be this what it may—not at the disk, but at some other part of the nerve or tracts—and which only shows itself after a shorter or a longer period at the nerve entrance as a secondary result.

Consecutive Atrophy.—This is the wasting away of the nervous elements, which follows the retrogressive stage of inflammation. When due to an inflammation which has affected the intraocular tissues, consecutive atrophy is subdivided into papillitic atrophy, or atrophy from retinitis or choroiditis, according as it is the result of inflammation in one or the other of these tissues.

Neuritic Atrophy.—One would suppose that this term would be applicable to that which followed neuritis, and especially to those forms seen with the ophthalmoscope. Leber, however, uses the term neuritic atrophy to express the results of an inflammation of the nerve-stem behind the eye, and of which there are either no or very slight signs of inflammation ever seen at the disk, or which, if they had ever existed, have entirely disappeared, leaving no traces behind. This is necessitated by the fact that he uses the term papillitis for all inflammations of the head of the nerve.

Ophthalmoscopic Appearances.—The two principal symptoms of optic-nerve atrophy as seen with the mirror are an increasing or increased whiteness of the disk and a reduction in the amount of the circulation. This latter, although less conspicuous than the former, is certainly earlier in its manifestations, and is indeed the principal cause of the latter—that is to say, the increasing whiteness of the disk is due rather to the absence of the rosy tint of the circulation than to any change in the condition of the nerve-fibres themselves, although, as will be seen immediately, there is a change in their physical or chemical condition which also contributes to the effect.

The first notable change which takes place is a reduction in the capillary circulation in the outer parts of the disk, probably simply because the vessels are less numerous here than in the nasal portion. As the result of this decrease in tint, the small vessels which leave the disk at the temporal side become more marked than in the normal condition through contrast with the whiter ground, which may, even at this early stage, begin to assume a slightly bluish tint.

Such a condition as this is often seen in some of those cases which have been spoken of as retro-bulbar neuritis, and which end in partial

atrophy. Indeed, not infrequently, I have seen a condition which would appear to be a mixture of congestion and atrophy at the same time—the temporal side showing the symptoms of atrophy, confirmed by a defect in the field, while the nasal side presented unmistakable signs of congestion. Not only have I seen such appearances in the atrophy which followed retro-bulbar neuritis, but also, not infrequently, in cases that would be pronounced as chronic glaucoma, but which I believe to be of a species of neuritis—that is to say, the result of a low stage of neuritis followed by excavation.

Little by little the rosy tint vanishes also from the nasal side, the concentric markings are lost, so that even the connective-tissue ring is hidden from view, or is only detected by very careful observation. Sometimes, however, when the central parts of the nerve are not as pallid as usual, the connective-tissue ring may appear, from contrast with a duller centre of the nerve, as whiter and more conspicuous than usual. The pallor then extends over the entire surface of the disk. The smaller vessels running toward the macula then disappear, although very often they persist for a considerable time. While this is going on, the larger vessels seem to participate but very little in the wasting away.

The arteries may have become a little narrowed and the veins even less so. Indeed, I have seen cases where the process seemed to be arrested with the total disappearance of the capillary circulation, and where for years there was no reduction in the size of the larger vessels, either arteries or veins.

These cases are usually those in which the offending cause is a temporary one, and which occur from some toxic condition, especially in those that have been attributed to uræmic poisoning, those from menstrual retention and uterine disorders, and particularly those incident to childbirth. The same thing may happen in spinal troubles, but to a lesser degree.

As a rule, it would seem that the more primary the atrophy was, and the greater the distance of the original lesion from the eye, the more apt the larger vessels were to maintain their calibre, and for a longer period. Just the reverse, however, may occur even in primary atrophy, and I have occasionally seen the larger vessels become smaller and smaller, while almost the natural tint of the disk was maintained.

This is apt to take place in that form of atrophy where abuse of the eyes on fine work, and poor illumination, seem to be the only causes of the wasting away.

Later in the trouble, as a rule, the larger vessels are considerably reduced in size, especially the arteries, until, in some cases, they as-

sume thread-like proportions; they are, however, never so reduced in size that they can not be much further traced into the retina, in the primary forms of atrophy, than where the atrophy is the result of inflammation of any degree, such as in papillitis or consecutive atrophy of the retina. In this case the vessels are not only smaller but are much less distinctly seen, partly because, owing to secondary contraction, they are really reduced to their ultimate limits of fineness, and partly because the neighboring tissue is less transparent, and thus partially conceals or dulls their outline.

In some cases of primary atrophy the light-streak, although lessened in proportion to the decrease in the vessel, is plainly seen, showing that the wall of the vessel is not implicated, while in other cases it disappears entirely, even when the process is comparatively recent and the vessels are still comparatively large. This would show that the walls of the vessels were also involved. Indeed, I have seen cases where I have been led to believe that the atrophic process was principally due to a low stage of vasculitis, or perhaps sclerosis of the wall, with gradual extinction of the smaller vessels, and consequent loss of nourishment to the tissue. This loss, or reduction of the light-streak, happens not infrequently in cases of amblyopia potatorum, and other amblyopic conditions of toxic origin; and sometimes also in cerebral affections. The effect is as if there had been a mild form of interstitial or medullary neuritis which had crept down the nerve and extended itself along the vessels, giving to their walls at times a greater visibility, or producing even fine white lines along their borders.

The whiteness of the nerve, as above related, already reduced to a predominating degree by the loss of the rosy tint caused by the absence of the vascular supply, is, it is said, increased also by some chemical or physical change in its fibres which increases their power of refraction. But, in admitting this, it should be borne in mind that the increased whiteness thus produced must be occasioned by the non-medullated portion of the fibres at or near the lamina cribrosa, where the fibres lose their medullary sheath; for, as long as those fibres which form the disk retain their transparency it would make no difference upon the picture presented by the mirror how much their index of refraction and power of reflection were increased. That their transparency is not affected is shown by the fact that, as a rule, not only is the reticulated structure of the lamina not obscured in primary atrophy but it is even more apparent than in the normal eye. The marbleized and talc-like appearance of the disk does not, therefore, come from the head of the nerve, but from the tissues behind it.

The whiteness of the atrophic nerve is also increased by contrast with the choroid and the clean-cut edge which it presents against this ground, as in primary atrophy; in contradistinction to some forms to be mentioned later, where the edges of the choroidal opening are frayed and absorbed either through inflammation or by pressure, as in glaucoma. On this account, too, the size of the nerve does not appear to be changed. Nevertheless, even under these conditions, it does occasionally appear to be reduced in size even in simple primary atrophy. On the contrary, in some eyes where there is a small crescentic cone at the outer edge, the surface of the nerve, to a cursory glance, seems to be enlarged. Close inspection, however, can always detect the fine line which marks the boundary of the real papilla and separates it from the sclerotic ring.

Again, the whiteness of the nerve is increased at times by increased cell development, or by the presence of connective tissue—that is to say, the place of the wasted and atrophied fibres is supplanted by connective tissue, which to a careful observer betrays its nature by its texture, by its softer tone, and more yellowish color; by the fact, too, that it usually covers more or less completely the vessels, at their point of juncture at the porus opticus, with feathery or veil-like expansions.

The condition of affairs would seem to justify, as suggested by Charcot and Abadie, a subdivision of primary atrophy into two classes—that which was dependent upon actual atrophy of the fibres themselves, and that which was occasioned by a hyperplasia of the interstitial tissue, by which the fibres were gradually supplanted. Such a division would appear to be in accordance also with clinical experience, for one not infrequently sees cases where the nerve has a marked degree of whiteness, and yet where the vision is disproportionately good—indeed, nearly normal. In these latter cases, of course, the fibres, or at least the larger part of them, have as yet, maintained their integrity, which no doubt will be eventually impaired by further contraction of the surrounding tissue.

It might be mentioned in this connection that I have occasionally seen disks which presented so high a degree of whiteness that it would at once have been pronounced pathological, but where no complaint had been raised as to any trouble of the eye, and where vision was, indeed, fairly good. I have looked upon these cases as the result of some low form of interstitial neuritis occurring very early in life, or perhaps congenitally, as in fact analogous to that form of retinitis which has been described as retinitis proliferans—in other words, a mild form of neuritis, in which the hyperplasia of the connective

tissue had only been sufficient to damage but not to destroy the nerve-fibres, and which had found its expression in an increased whiteness of the disk.

Gray Discoloration, or Gray Atrophy.—This occurs not infrequently as the result of atrophy. Here the disk, instead of being of a uniform and rather brilliant white, has a distinctly grayish tinge, which is usually most marked near the outer or temporal side of the disk, especially in the beginning of the trouble. It is often sickle-shaped, with a space between its outer border and the edges of the nerve. When this occurs it has precisely the appearance as if the nerve had been shaded by art to produce the effect of an excavation, which is then sometimes so strongly and accurately figured as to lead even a practiced observer to believe that there is really an excavation where none exists. A second sickle-shaped formation may also be present at the inner side, or the color may extend in a zone round the nerve at a little distance from its edge. Sometimes, again, the color assumes a sector-like character, with fine white radiating lines between the fan-like divisions. On the other hand, the color, especially in the older cases, may extend uniformly over the entire disk. In these cases the connective tissue may assume a conspicuous appearance, and seems to surround the head of the nerve as the tire does the wheel. Or, still again, the surface of the nerve may have a mottled, gray appearance, and when there is an excavation combined with it the darker spaces, seen at the lamina cribrosa in normal eyes are rendered still darker (see Jaeger, "Hand Atlas," Figs. 48–51). To what the gray color is due is not known. Under the microscope the nerve-tissue has a translucent, with perhaps a slight grayish tinge to it.

It is claimed that slight variations in color, especially the beginning whiteness of the nerve, is better seen with the Helmholtz mirror than with the silvered one, as the light is more subdued. So far as the slighter variations of the greenish and bluish colors are concerned, the weak light mirror is certainly of advantage; but it has never struck the writer that, for the detection of the first traces of increasing whiteness of the disk it offers any advantage over the ordinary concave mirror. Indeed, I have often thought that it was through the very excess of light that I have been able to detect the earlier stages of atrophy; that, in other words, the increased illumination brought out the beginning whiteness more than it quenched the rosy tinge of the now atrophic parts, so that there was a greater contrast between the two. I have therefore often used advantageously the inverted image for this purpose.

Bluish Discoloration of the Disk.—Besides the general white or gray color which extends over the surface of the papilla, there is a peripheric change in color which is usually of a pale-bluish, or skim-milk color, but which at times may be increased to a light but pure blue.* This peripheric and bluish discoloration occurs most frequently at the outer border of the nerve, but may extend round its entire circumference. It is sharply limited from the connective-tissue ring and extends gradually, becoming paler as it proceeds, toward the centre of the nerve, which then may be of a dull-grayish color. It is commonly met with in spinal and cerebral troubles, and after chronic inflammatory conditions of the nerve and retina.

Green Discoloration.—These in their lighter shades are not infrequently seen at the periphery of large and deep pathological excavations of the optic nerve. It is occasionally so well marked as to have a very perceptible green color, and in extremely rare cases a bright emerald hue, such as Jaeger has pictured in his Atlas, and which for a long time I believed to be marked exaggerations. I have, however, seen one case which fell but little, if at all, short of the tint depicted by Jaeger (see frontispiece, Vol. II).

None of these discolorations would appear to be due to any coloring matter in the tissue. It would certainly, from the autopsies hitherto obtained, appear that the gray color was not due to any collection of pigment. They are all probably due to varied reflections of light, and minute differences in level with interference of the rays.

Discoloration of the circumference of the nerve, and the neighboring pigment layer of the retina, may take place in old people, producing a white concentric figure, and has then a strong resemblance to posterior staphyloma. When simply the result of a slow wasting away of the tissue, it does not result in any loss of vision, but at most only increases the size of the blind spot. I have, however, seen two cases in which the appearances were precisely like those detailed above, with the exception that the apparently large staphyloma extended round the entire nerve, and was of a yellower color than the ordinary cone. The trouble came on rather suddenly in each case, and vision, bad in both, in one case was affected almost to complete blindness; the refraction as measured by the ophthalmoscope was emmetropic.

It will be well to remind the reader in this connection that peculiar forms of injection may occur as congenital anomalies which give a spotted character to the nerve, or that there may be small places in the surface of the disk which differ in hue and brilliancy from the

* Jaeger's "Hand Atlas," Figs. 45, 46.

rest of the papilla. Thus, I have seen a case in which the temporal side of the nerve seemed to be made up of small circles of about the size of the smallest seed-pearls. It is important not to confound these and other such variations of a congenital and physiological character with the discoloration which arises from disease.

Atrophic Excavation of the Nerve.—This is characterized by a slight depression of the surface of the disk, which is especially marked ward the temporal side, and that extends, as does a glaucomatous cavation, up to the edges of the nerve. As a rule, however, it is easily distinguished from this by its shallowness, and from the fact that the vessels are but very little, if at all, displaced from their course, and show no curving over the edges of a crater-like opening, as they do in glaucoma.

Sometimes, however, an atrophic excavation may be grafted upon an abnormally large physiological excavation, and here it is very difficult, if not impossible, to say whether in a given case the excavation is atrophic or glaucomatous, or a mixture of both. Still, even here the behavior of the vessels, the absence of the effects of increased intraocular pressure upon their course and conformation, enables us to discriminate between the two; as does also the fact that we can usually see that the floor of the original physiological excavation is on a different and more posterior plane.

I might say here that I am convinced that an atrophic excavation is not nearly as frequently met with ophthalmoscopically as it is microscopically; for, although a depression does occur in the wasting away of the tissue, this is comparatively light in itself except in exceptional cases, and is in great part counterbalanced by the fact that the nerve-fibre layer and ganglionic layer are also atrophied and reduced in thickness, which reduces the general level of the retina. Moreover, we see the head of the nerve directly "on," and not in section as with the microscope, and we have no means of judging with the mirror such slight depressions as would be apparent to the microscope.

Again, I have not infrequently convinced myself that what I, as well as others, had at first sight taken for an excavation was indeed no excavation, but, for reasons just narrated under gray discoloration, a pure optical delusion due to the coloration of the disk and the effects of light and shade, which are precisely those which an artist would employ if he wished to produce the appearance of an excavated nerve upon a flat surface like paper.

This happens especially in those cases where there is a bluish or grayish discoloration, which then appears to be graduated in tone so as to produce the sensation of a concavity. I mention this because

the detection of the true condition has prevented more than once the performance of double iridectomy for chronic glaucoma. An atrophic excavation, as has already been stated, has a greater tendency to show color than does a non-excavated but atrophic disk, and this is particularly marked in the mixed atrophic and glaucomatous excavations.

Such being some of the principal features of primary atrophy, we pass now to a consideration of the changes produced by secondary influences.

Secondary Atrophy.—This is the form of atrophy which, as has already been explained, results from some lesion, the original seat of which is posterior to the bulb, while its manifestations are shown at the disk in a secondary manner in the shape of atrophy of the nerve. When this is due to direct pressure upon some portion of the extra-ocular part of the optic nerve from some growth, the atrophy which follows and becomes visible at the disk bears so striking a resemblance to primary atrophy that it is frequently difficult, sometimes impossible, to tell the difference between the two. All the more so when the atrophy has been a long time in reaching the disk, and has pursued a slow and chronic course after having reached there. One, and perhaps the most distinctive, mark, and one which often continues for a long time, is the fact that the capillary circulation seems to be principally affected, while the larger vessels appear to have undergone little or no change, either as to calibre or course. This is especially true of the veins. At a later period the larger vessels begin to diminish in size, the arteries first and then the veins.

Secondary atrophy which is not the result of direct pressure, but which is due to inflammation of the nerve-stem or its sheaths, is like that due to atrophy which follows interstitial neuritis where there is hypertrophy of the connective tissue. Here the disk is less white and brilliant in hue and has a more cottony look, and has occasionally a grayish or dirty appearance. The larger vessels usually begin to grow narrower even before the capillary circulation has disappeared, and, as the process continues, they rapidly diminish in size until they at times become thread-like. The vessels also are bordered with fine white lines and show particles of feathery tissue at the porus opticus, and the light-streak upon the vessels is either very much diminished or lost. Indeed, in some cases one is in doubt whether he is in the presence of a subsiding interstitial neuritis or a beginning atrophy. The state of the vision and field may coincide with that of atrophy, and yet the disk, while it shows pallor and discoloration in the outer parts, may present some signs of congestion in the nasal portions.

Later on, however, when the atrophy has become more marked, the distinction is made at once.

It is hard to account for the reduction of the vessels in these cases. The supposition that it is due to contraction of the tissue from retrobulbar neuritis would not appear to be sufficient, as oftentimes the focus of inflammation is situated too far back to have any effect in constricting the vessels in the scleral opening, or as they enter or leave the nerve, especially as it has been shown conclusively that very little if any constriction takes place at the scleral opening, even in the more violent forms of choked disk and perineuritis. The narrowing of the vessels is probably due to some morbid condition of the walls of the vessels themselves by which their transparency is diminished and their calibre decreased.

Secondary atrophy visible at the disk not infrequently follows inflammation of the sheaths, or perineuritis, which has originated in some orbital process, such as cellulitis of the orbit from whatever cause, in which is included erysipelas of the face. Tumor of the orbit, however, usually occasions intra-ocular neuritis, such as choked disk, and the atrophy which follows is rather consecutive than secondary.

Consecutive Atrophy.—This title is used to designate the result of some preceding intra-ocular inflammation. Its characteristics have already been described in part under the "retrogressive changes" which follow papillitis, retinitis, and choroiditis, in their various forms. It will, therefore, only be necessary to remind the reader that "papillitic" atrophy is one that affects the head of the nerve without affecting the stem or implicating the retina, and which leads to atrophy of the intra-ocular end of the nerve. The principal features of this form of atrophy are a filled-in and more woolly aspect of the disk, with increase of the connective-tissue elements. There is marked narrowing of the vessels, especially the arteries, which can sometimes only be followed for a short distance into the retina. There are signs also of perivasculitis. The color of the disk is a dull and not a brilliant white, and often has a dirty grayish appearance. The close fibrous and reticulated connective tissue into which the papilla is changed covers the lamina cribrosa, and not infrequently conceals the choroidal border. In the beginning the papilla is somewhat prominent, but later becomes flatter, so that the normal level is finally maintained. The borders of the disk are not sharply defined, as in primary atrophy, and the papilla appears to be somewhat larger in size, since the edges of the choroid are overlaid by it. The disk may also show the remains of fatty degeneration and sclerosis.

For a further consideration of these matters I would refer the reader to Chapters I and V, and to the general remarks upon atrophy of the central vessels.

Retinal Atrophy.—Atrophy of the optic nerve from retinal disease, such as retinitis pigmentosa, chorio-retinitis, and other chronic forms of retinal inflammation, is chiefly distinguished by the fact that the color of the papilla is never of that clear and brilliant white, or bluish white, that marks primary or extra-ocular atrophy. The tone is more opaque, and of a yellowish-gray color, which extends more uniformly over the entire surface of the disk. There is less tendency to excavation, as the nervous elements are supplemented by an increased development of the interstitial elements. The edges of the nerve are uneven, and apt to be dotted here and there with small masses of pigment, which, when found in the retina itself, show the true origin of the atrophy. The vessels are apt, as after all inflammatory processes of the retina, to be exceedingly narrow, and to show in a greater or lesser degree the signs of vasculitis.

In the more acute kinds of retinitis, such as retinitis albuminurica and hæmorrhagica, the signs of atrophy are apt to resemble those of the higher grades of neuritis and neuro-retinitis, such as have been described under consecutive atrophy and the retrogressive changes in choked disk, where there are grave disturbances in the circulation, and even venous thrombosis.

Microscopical Characteristics of Atrophy of the Optic Nerve.—In the earlier stages of atrophy there is usually but little reduction in the size of the nerve, though at times, however, there may be some, but it has a transparent aspect, and a softened and friable consistency. The atrophic nature of the trouble consists in the decay of all the elements of the nerve, with the results of the degeneration of the fibres, globules of fat, granular cells, and amyloid bodies. When interstitial inflammation has been present, there is usually an increase in all the connective-tissue elements, which seem then to have supplanted the nerve-fibres. When the atrophy has been the result of pressure, especially that from perineuritis through contraction, the nerve is much reduced in size, and at times appears to be changed into a cord of connective tissue.

In gray degeneration of the nerve there is an increase in the connective-tissue staging work, with an atrophy of the nervous fibres, either with or without a previous development of the granular cells. The atrophied nerve-bundles become reduced in size and of irregular dimensions.

The atrophy may be more extensive in some portions of the tis-

sue and also in some areas of the same section, being more marked near the periphery, as a rule, although at times it may be more developed in the axial portion, and close to the vessels. The medullary substance is first affected. By the disappearance of this and by the reduction in the diameter of the fibre the size of the nerve is also reduced. By the loss of the medullary substance the fibres become non-medullary fibres and grow varicose. Later these varicosities disappear and the fibres then appear as fine homogenous fibrillæ. With these changes the nerve assumes a gray, translucent, and jelly-like appearance. According to Gowers, a gelatinous substance is also found round the vessel which contains a few nuclei and an indistinct concentric fibrillation.

In papillitic atrophy, or that which is the result of an inflammation of the head of the nerve, there is a great increase in the connective-tissue fibres which, here particularly, supplant the nervous fibres either to a great degree or even entirely. The surrounding retina shows distortion and displacement of its elements, and the shape of the choroidal opening is changed.

Visual Defects.—One of the most important symptoms of atrophy of the optic nerve is a disturbance in the amount and character of the vision. This usually begins with a peripheric and concentric narrowing of the field of vision; and, as this form of limitation is different from what occurs in what may be called the benign amblyopias, it forms an important element in the diagnosis. The limitation may begin at any part of the periphery of the field, the point of selection varying according to different authors. Its great characteristic is that it is concentric, and has usually advanced some way, before central vision begins to decline; so that the observer, whenever there is a suspicion of blanching of the disk, should accurately test, not only the acuity of central vision, but also that of the periphery of the field.

There may be occasionally, besides the concentric narrowing, and occasionally without it, patches of defect which then form scotoma. A pure central scotoma in simple progressive atrophy, without concentric narrowing, is very rare in the earlier stages of the disease, yet even this may occur. In axial or partial atrophy a central scotoma is the rule, as has been pointed out, and this may then be for both perception and color. Fig. 55 shows the concentric limitation of the field in an ordinary case of progressive atrophy.

In some cases the field in both eyes is so limited as to resemble what occurs in hemianopsia—that is to say, the same side of each eye, either the right or left, is affected. Sometimes, however, although

much more rarely, the opposite is the case: thus I have seen several times nasal hemiopia where the inner half of each retina was affected; and what at first sight seemed to be a limitation, due to cerebral disease, proved later to be caused by simple progressive atrophy. Fig. 56 shows such a field, in which the nasal side of each retina was affected. The shaded portion denotes the part of the field that is wanting.

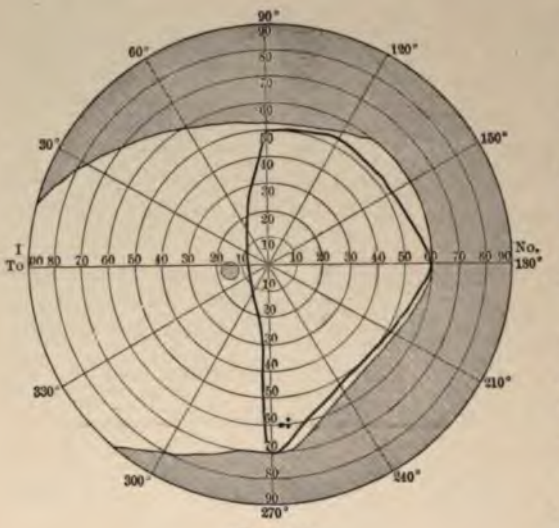

FIG. 55.

As a great rarity the blind spot alone is enlarged. Thus, I once saw a case in which the blind spot was so enlarged as to extend nearly up to the point of fixation in both eyes.

Color Disturbances.—In all kinds of atrophy there is a confusion of colors, even in the earlier stages. Green is confounded with gray, or at times with yellow, while rose and purple appear bluish. The perception of different colors behaves in atrophy as it does in the normal eye—that is to say, if the colors green, red, blue, yellow, and white are carried from the point of fixation toward the periphery, the sense of green will be lost first, followed by red, blue, yellow, and white, in the order named.

Fig. 57, from Gowers,* represents the normal field for color, and in atrophy, as in health, the colors are lost in the order in which they are named. There is, though, no constant proportion between the degree of the disturbance in color and the amount of amblyopia. Sometimes, however, the loss of the sense of color points to the fact of beginning trouble, which is afterward confirmed by a loss in the field.

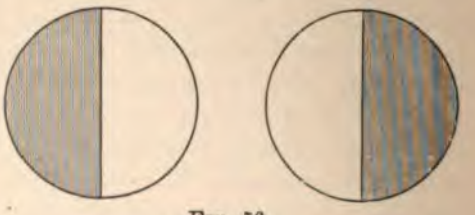

FIG. 56.

* "Medical Ophthalmoscopy," p. 104.

Causes of Atrophy.—Simple atrophy occurs in a great many cases in a perfectly independent manner, and where there are no signs of any affections of other nerves, or indeed of any complication of any disease whatsoever. Primary atrophy is much more frequent in men than in women, and in adult life than in childhood. Papillitic, or consecutive atrophy, on the other hand, is equally as frequent with women as men, which may be due to the fact that, besides being subject to syphilitic disease, they are exposed during gestation to kidney trouble and associated neuro-retinitis. In partial atrophy men are much more affected than women —due, no doubt, to the greater influence of tobacco and spirits. Hereditary atrophy is also more common with men than women.

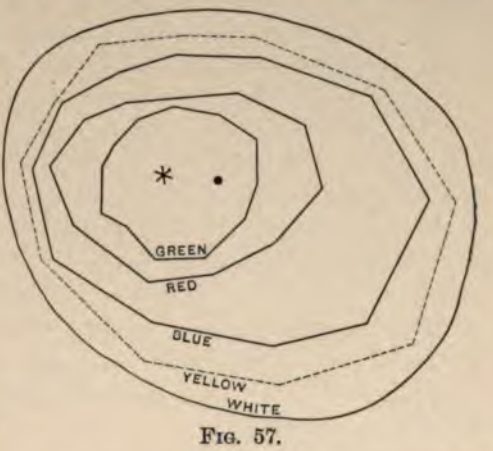

FIG. 57.

It may also be stated, in general terms, that wherever neuritis has occurred, or pressure been exercised upon any part of the nerve, atrophy may follow. This alone would make the etiology of the disease as broad and general as the list of diseases themselves, and hardly possible, even if worth while, to enumerate—especially as so much has already been said in regard to neuritis. So often, however, does atrophy occur with spinal and cerebral diseases, that it is sometimes, in a way of designation, convenient to speak of a "spinal" or "cerebral" atrophy of the optic nerve. Atrophy of the optic nerve in spinal troubles depends, as does the disease of the spine itself, upon gray degeneration of its nervous tissue. In rare cases, however, the nerve to the mirror is not gray but white, and even of a lustrous hue.

The gray degeneration does not limit itself to the nerve-stem, but attacks also the chiasm, and extends back to the corpora geniculata, if not farther, as modern investigators are inclined to believe. Gray atrophy usually affects both eyes, although often, if not usually, in very different degrees, both as to the time of the appearance of the ophthalmoscopic signs and the state of the vision. Thus several years may elapse before the second eye shows any changes either in vision or in the appearance of the nerve. Indeed, one eye may advance to total blindness while the other remains entirely normal. In rare

cases it is said that only one eye is affected. The disease may have remissions for a longer or shorter period, and vision may appear to improve for a time, only to begin to fail again on some imprudence, or some reduction of the general health, or from no apparent cause. As a rule, however, the disease runs its course, in from one to two years, when blindess ensues.

In atrophy from spinal disease the diagnosis is often helped by the detection of muscular troubles, the shape of the pupil, and the presence of rheumatic and ataxic pains, paresis of the bladder, and the characteristic gait, as these usually precede the eye symptoms. Still, this is not always so, and it is often by means of the mirror that the spinal trouble is first detected or suspected.

Atrophy of the optic nerve may occur in myelitis of the lateral columns. It may also occur after injuries to the spine, but only after a considerable lapse of time. In recent cases, according to Allbutt, only hyperæmia of the disk is usually found. This may, however, be associated with obscured borders of the nerve, moderate tortuosity of the veins, with, at times, a considerable decrease in vision. These facts would suggest rather some form of commotio-retinæ than neuritis from meningeal trouble, as suggested by Allbutt, especially as the case had a tendency toward convalescence rather than toward atrophy of the nerve.

Atrophy from Cerebral Disease.—The use of the word atrophy in this connection commonly excludes all those causes which produce intra-ocular inflammation, such as neuritis and choked disk, and applies only to the atrophy that happens to follow direct pressure upon the nerve-stem. This may happen from tumors or growths, such as tubercle and gummata. It may also occur from hydrocephalus internus or hypertrophy in the region of the chiasm or at the optic foramen, including exostosis of this and other regions, and aneurismal dilatations and constrictions of the optic nerves by diseased vessels. It will be remembered, of course, that most if not all of these do produce intra-ocular inflammations, but occasionally do not, and it is under such conditions that the name of cerebral atrophy has been applied.

Strictly speaking, the atrophy that accompanies one-sided brain-disease, and which is expressed by hemianopsia, ought to be included among the cerebral atrophies, and no doubt would if it showed itself more frequently at the disk; but it requires so long a time for the atrophy to descend to the disk, that it very rarely manifests itself to the mirror. In the few cases yet reported there seems to have been a difference in the appearances noted, since in some cases there were

the signs of atrophy extending over the entire surface of the disk on the side opposite the lesion, and in other cases one half of each nerve was affected.

Sometimes we find gray degeneration of the nerve, with softening, as we do in disseminated sclerosis of the brain and progressive paralysis of the insane. In sclerosis of the brain, paralysis of the muscles of the eye is usually combined with the cerebral trouble. There may be also dilatation of one or both pupils, and at times nystagmus. In general paresis simple atrophy often occurs, and is often, as before stated, preceded by a congestive stage of a light character. The affection of the eye, unlike that in spinal disease, frequently precedes the motor disturbance.

Atrophy of the Optic Nerve from Orbital Disease.—This may occur as the result of affections of the orbit, which exert pressure either directly or indirectly upon the nerve. The atrophy may take place at the disk without any signs of previous inflammation having occurred, although such may have been present at some time previous to the examination.

Glaucomatous Excavation.—A glaucomatous excavation is one which signifies that the head of the nerve has been subjected to a pressure upon its surface, which is greater than it can bear, or, in other words, that the force applied is greater than the resistance. Thus an excavation may result under different conditions: for example, the intra-ocular pressure may be increased over the normal amount, and the power of resistance be normal, as usually happens in acute glaucoma; or the intra-ocular pressure may not be above the normal, and the power of resistance of the tissues of the head of the nerve be so much reduced that they yield even to a normal amount of intra-ocular tension, as happens usually in glaucoma simplex; or, again, the intra-ocular tension may be less than the normal, and the power of resistance still less—by which it happens that we often see enormous excavations of a purely glaucomatous type in eyes in which the intra-ocular tension is below the normal standard, and in some cases to a marked degree.

The chief characteristics of a glaucomatous excavation are its extent, its depth, and the behavior of its central vessels. Its enlarged size depends upon the facts that it extends up to the edges of the scleral opening, and that, as this is beveled backward, a sharp edge is presented to the observer, and an opening is thus produced with overhanging, counter-like borders (Fig. 58). In other fully formed cases the excavation extends up to the edges of the nerve for its entire circumference, but it may happen in the earlier stages that it

does so only in portions of the circumference, and in these cases it is usually, though not always, at the temporal side. Under these conditions, which may be looked upon as a sort of partial excavation, the excavation is not a deep one, though the opposite may be true, and when it is, the central vessels are usually crowded toward the nasal side. It is sometimes difficult, then, to say whether the cupping is a commencing glaucomatous excavation, or a physiological one, or a mixture of both—that is to say, a physiological being transformed into a glaucomatous one.

The depth of the excavation depends upon the fact that the tissue of the lamina cribrosa is at first pushed backwards by the

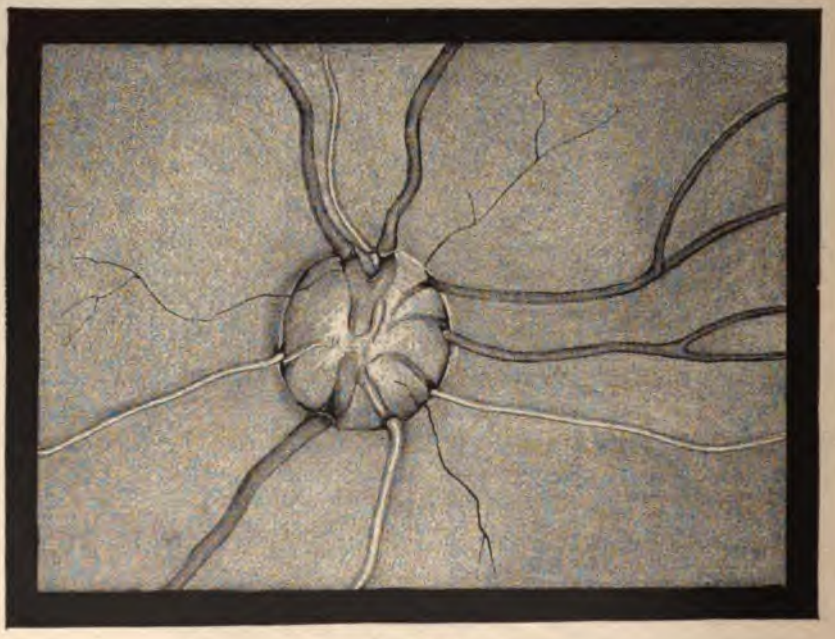

FIG. 58.

pressure upon it. In the earlier stages, the anterior portion of the lamina is concaved in front, while the posterior border maintains its original position; later this also gives way, and the tissue in front of the lamina is absorbed until only the merest trace of it is shown as a membranous expansion at the bottom of the excavation. It is this implication of the lamina and the changes that it undergoes which chiefly distinguish a glaucomatous, from a physiological or atrophic excavation, in which the lamina retains its normal plane,

or varies only slightly from it. The depth of a glaucomatous excavation may vary from one millimetre to nearly two millimetres, as measured from the inner surface of the edge of the nerve by the ophthalmoscope. The method by which these measurements are made has been shown (Vol. I., p. 115). The floor of the excavation

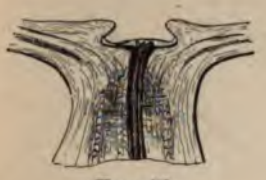

FIG. 59.

FIG. 60.

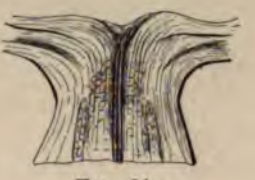

FIG. 61.

may be of different shapes and of different levels. Thus it may be flat and broad, as in Fig. 59, where the section of the excavation has the shape of a truncated pyramid, or it may be cup-shaped as in Fig. 60, or in the form of a funnel, as in Fig. 61.

Ophthalmoscopic Appearances.—These, for the sake of convenience, may be divided into those accompanying the initial and the advanced stages, and those which are characteristic of chronic (or simple) and acute glaucoma. In the earlier stages of simple glaucoma the disk may preserve to a considerable degree its normal injection, and the difference in level may be so slight as to easily escape notice. When present it is usually detected by a slight bending of the vessels at the temporal and lower portion of the nerve. There is at this stage no disconnection of the course of the vessels, which can be followed even by the upright image up to the point of emergence. It is, of course, very difficult to tell at this stage whether the slight depression existing is due to pathological causes or not, and it is also difficult to determine whether the injection of the disk, which may be at times quite marked is physiological, or pathological and not due to a low form of neuritis. Indeed, I am more and more convinced that the initial step in glaucoma simplex is often a low state of inflammation, and that the excavation which follows is due to the reduction in the resisting power of the lamina and surrounding tissues, which has been lessened by the mild form of neuritis. Thus I have not infrequently seen a well-marked glaucomatous excavation on one side, and neuritis of a mild but still distinct form, with stretching of the tissue and a beginning excavation in the other eye, and where great reduction of vision in the more advanced eye, with lesser reduction and limitation of the field in the other, pointed conclu-

sively to the nature of the trouble. It must be borne in mind here, however, that the tension of the eye in chronic glaucoma may vary from day to day, or even from hour to hour, so that at one time it may be above the normal and again below, without any appreciable difference in the ophthalmoscopic signs, although the increase is apt to be accompanied by an increased disturbance in vision. In these cases I believe, therefore, that it is not so much an abnormal increase of the intra-ocular tension as it is decrease in the power of resistance in the head of the nerve, and further for this very reason, I am of the opinion that the same low stage of inflammation, or neuritis, which may lead in one case to simple atrophy of the head of the nerve, where a normal resistance is maintained, may at others lead to a marked glaucomatous excavation, where it is reduced. In this connection it is well to remember that in acute glaucoma the excavation is not usually developed by a first attack—that is to say, when the first attack has passed away, and the fundus can be viewed with the ophthalmoscope, there are, as a rule, no signs of an excavation. These may follow later and often do, even after a few attacks or even a single one, which shows that it is the want of resistance produced by the acute stage, and not the increased pressure at the moment when this is the most marked. Of course, it will be understood that there are other cases of a more chronic form, where the pressure is constantly in excess, and where the excavation is apparently due to the heightened pressure alone. But even here the initial step may have been due to an inflammatory process. As the process progresses the excavation increases in depth, the injection of the disk becomes less, and may assume even a normal whiteness, but never until the later stages, that of atrophy. The arteries become narrower and the veins more dilated, as if the blood, from the pressure exerted upon it, and from the resistance it encountered from the excavated shape of the disk, had a greater difficulty in entering the arteries and also in leaving the veins. In this stage, and even in some of the later ones, the excavation may be the seat of an increased injection, and on one or two occasions I have seen the entire floor of the excavation crowded either by what was a hæmorrhage, or, as I sometimes convinced myself, was a congeries of minute vessels, so fine that it was with the greatest difficulty that the single vessels could be detected. These cases are usually associated with tortuosity of the larger vessels. At this time the yellowish-white ring, so characteristic of glaucoma, begins to make its appearance, at first in segments and then as a complete circle. The outer border of the ring then often appears fuzzy and is extended irreg-

ularly out into the choroid. When the pressure is marked, the arteries have often the peculiarity of maintaining their normal size in the neighborhood of the disk and rapidly diminishing in calibre toward the periphery, so that at times they can only be followed for comparatively short distances into the retina. Another characteristic symptom, though by no means a constant one, is the pulsation, not only of the veins, but also of the arteries; when not spontaneous, it can be readily produced by the slightest pressure, and the ease with which it is produced then gives some indication as to the abnormal increase of the intra-ocular tension. Contrary to what holds in the normal eye, it is produced more easily in the arteries than in the veins, and a moderate pressure seems to produce it to a greater degree than a too forcible one, which may, indeed, cause it to cease.

When spontaneous, it is more noticeable in the arteries than in the veins, and seems to be quicker in action, and it extends for quite a distance, at times out into the retina. It may be either constant or intermittent, and cease entirely for a time, to reappear, at another period, either hourly, daily, or at longer intervals. It is more apparent at the period of access than remission, and more noticeable during temporary aberrations of vision than when the sight is at its best. In the stage of complete development, the ophthalmoscopic picture, by the direct method, is a very marked one, and needs but little verbal explanation; and yet it is precisely in this stage that the true nature of the affection was, in the early days of ophthalmology, totally misconstrued, and what was, in fact, a high degree of excavation, was taken for a marked degree of prominence of the head of the nerve. Knowing that it was an excavation, I have never been able to get the effect found and figured by the earlier writers, especially Jaeger, but as a curiosity it is certainly worth mentioning. As a rule, the vessels advance with but little noteworthy change to the edge of the nerve, where they are seen to make a sudden curve, which is marked by a darker color. They then are lost to view on the surface of the disk itself, or become of so pale a hue as to be barely recognizable, nor can the veins be told from the arteries. Moreover, when thus seen at the floor of the excavation they seem to be disconnected from those which come from the rim of the nerve, and dropped, as it might be expressed, "endwise." Sharp definition ceases with the rim of the excavation, and everything at the surface of the disk appears indistinct and hazy, and it is only as the correcting glass is made stronger and stronger that the details of the excavation come into view, while the edges of the excavation become proportionately indistinct. With the proper correcting glass the reticulated structure of the lamina

comes out, the difference between arteries and veins becomes apparent, and by slight movements of the observed and observer's eye, the vessels can be traced not only over the floor, but with occasional exceptions up its sides. This is not always so, however, as in some cases the vessels are so displaced sidewise and the excavation so undermines the edges of the long and deep tunnel-like pit, that the vessels remain concealed no matter what efforts are made to bring them into view by movements upon the part of the observed and observing eye.

Moreover, parallactic displacements of the vessels are obtained on the movements of the observer's head. The excursions of the pulsating vessels come clearly into view, and in some cases they seem fairly to leap from their bed. With a reduced light the pale-blue color, so often seen in glaucomatous excavations, comes into view, and in rarer cases the pale, and in still rarer, the emerald-green circle, especially at the temporal side, can be plainly seen. This last in its most conspicuous aspect is exceedingly rare, and I have only seen one single case which thoroughly realized in depth of color and brilliancy the drawings of Jaeger ("Hand Atlas," Taf. XI., XII.). Besides the changes at the surface of the disk, other changes occur in the retina proper, and chiefly in the vessels. These, as has already been pointed out, may on some occasions be very tortuous, and their walls may show signs of being affected. Nettleship reports a remarkable case, in which, besides all the well-marked symptoms of glaucomatous excavation, there was perivasculitis of a very high degree.

The choroid, too, in advanced cases is almost always affected. The white ring, which surrounds the nerve, is due to the atrophy of the choroidal tissue, in the part which is then reduced to a thin membranous expansion. This ring varies in width, and may extend, especially toward the macula, for a considerable distance from the borders of the nerve. The fundus, taken as a whole, has a faded, washed-out appearance, with occasionally more or less disturbance in the pigment layer. It is asserted that complications in the vitreous with opacities are not nearly so common as is supposed, and that the haziness of the fundus during the acuter stages is due to a want of transparency of the cornea; and that this is true in the main can be shown by the fact that the haziness of the membrane can often be detected by oblique light. Still, there are cases in which I have convinced myself that there were opacities in the vitreous itself, as would, indeed, be natural, whenever the uveal tract was affected. Again, it is probable that the colored rings seen round lights are due, not to opacities in the vitreous, but to want of transparency in the cornea, as sometimes occurs in catarrhal affections on suddenly wakening from sleep, where

the media are entirely clear and vision perfect. In this latter case, it is evidently due to small particles of mucus adhering to the surface of the cornea, by which on sudden exposure to a lighted lamp an interference of the rays is produced on their way to the retina. When the eyelids in their action have swept away these particles, the colored rings in these cases immediately disappear. With the inverted image, the picture differs somewhat. Here, as elsewhere, the variations in level do not make so much difference, and in the aërial reduced picture formed by the object-lens we obtain a more connected view, and one that is but little disturbed in its antero-posterior relations. Thus we see to the bottom of the excavation, and consequently, except in occasional cases, can trace the vessels in their continuous route over the edge of the disk and up to their emergence to and from the bottom of the excavation. The whole picture is clearer and brighter, but not so characteristic, and slight degrees of excavation are apt to be overlooked when this method is employed. I have sometimes thought, however, that slight pulsations were, whether spontaneous or produced, more easily detected from the very concentration and brilliancy of the picture, with the inverted than by the direct method.

Visual Disturbances.—It is very important to note these, especially in doubtful cases. Vision is usually affected early in the disease, and an examination of this is often the deciding point in the diagnosis. The deterioration of sight takes place principally in three ways—by rather sudden and temporary obscurations, by decrease of the central acuteness, and by limitation of the field. Central scotoma may occur here, as in atrophy, when certain fibres are affected or cease to functuate, but the usual form is a peripheric lessening of the field. There are some very exceptional cases, where there are all the signs of a marked glaucomatous excavation, with no reduction in sight whatever, either in acuteness or extent of field. These cases are probably those in which an enormous physiological excavation originally existed (such as is figured in Vol. I, p. 60), and on top of which is grafted a glaucomatous excavation, which is really in its earliest stages, from which the nerve-fibres have as yet suffered but little or nothing, notwithstanding the pronounced picture produced. How early the choroidal tissue can be absorbed in the neighborhood of the nerve without impairing the vision at all, is seen in cases of progressive myopia, in which the cone gets larger and larger, and yet vision remains unchanged or even normal. This would account for the atrophic ring surrounding the head of the nerve. Glaucoma is essentially a disease of middle or advanced life, but it may occur in youth or even childhood. Thus I have seen a case of excessive

glaucomatous excavation in a child of eight years. There was in my case a small anterior synechia, which probably produced the disease through increased tension. Causes of pure glaucoma simplex with excavation have been reported in children by Dr. Webster, and by Dr. Randolph, in which case the child was only eleven ("Archives of Ophthalmology," XVI., No. 2, p. 185). In buphthalmos and keratoglobus glaucomatous excavation may occur in very early life.

Finally, the reader must bear in mind that excavation of the optic nerve of a glaucomatous character may occur as a concomitant or sequel to almost any disease whatever, in which the pressure upon the head of the nerve is increased or its resistance lessened.

Pigmentation of the Nerve.—Cases of pathological pigmentation of the optic nerve have been reported by Liebreich, Knapp, and others, with the conviction that they were due to pathological and not to physiological processes, examples of which have already been mentioned (Vol. I., p. 89). Liebreich's case occurred in two atrophic disks in such a way that, while the borders of the disk were pigmented, the centres remained clear. It has been supposed that the pigmentation was due to hæmorrhage. I have myself at various times seen small masses of pigment in eyes which showed retrogressive and atrophic changes, but I never could determine that the small amounts of coloring matter were not physiological or congenital. Mauthner also mentions having on several occasions seen the disk studded with minute points of chlolesterine, in cases where the papilla had undergone pathological changes.

CHAPTER VII.

AFFECTIONS OF THE CHOROID.

Posterior Staphyloma.—This name has been applied to a distention of the membranes of the eye, by which principally the longitudinal axis is increased, and with it that of the refraction of the eye. This gradual increase in the longitudinal diameter of the eye is usually accompanied with a peculiar and striking appearance at the outer margin of the nerve, which has been variously designated, both on account of its form and supposed pathological origin. Thus, from its form, it is often called the "crescent," or, with little apparent reason, a "cone," or more frequently from its supposed origin, a posterior staphyloma. This latter name was given to it from the belief, in the early days of ophthalmoscopy, that there was an actual protrusion backward of the sclera and other membranes, so that a real ectasis existed. That this does occur at times can not be denied, as the existence of such protrusion can not only be determined with the mirror, but has been proved to exist, from early times, through anatomical examination.

Although the "cone" may be met with in early infancy as a great rarity, it usually does not make its appearance before the fourth or sixth year, and more frequently still somewhat later, about the tenth or twelfth year. The first trace of beginning "crescent" is not, as usually stated, in the choroid at the outer edge of the nerve, but on the surface of the disk itself. This consists of a gradual lessening of the capillary circulation, at the temporal side of the disk, so that the nerve at this point presents an abnormally white appearance, and through contrast the small isolated vessels, which leave the disk and run toward the macula, attain an undue prominence. The neighboring choroid then begins to show evidences of being affected. The tissue immediately surrounding the outer edge of the nerve begins to show for a little space a thinning, or, if the term may be used, a sort of rarefaction, in which the edges of the choroidal opening begin to look frayed, and the tissue more or less mottled over the future

crescent. Sometimes the pigment is increased, so that the border of the nerve is surrounded by a black line, like the heavy downward stroke of a pen, and this development of pigment may advance with the increase of the staphyloma, while the tissue is absorbed behind it, much as a ringworm advances over the skin. In this case the coloring matter usually becomes less thick and dense, as the crescent advances toward the macula, until sometimes it entirely disappears. On the other hand, the pigment may cover the entire staphyloma, and then forms a bluish crescent which may remain for a very long time, if not permanently (Jaeger, "Hand Atlas," Taf. XXVI.). In other cases, the small masses of pigment may be scattered over the general surface of the staphyloma, and be gradually absorbed in one place to make their appearance in another. On the other hand, a crescent may be formed of large size, with hardly a perceptible trace of pigmentation from the beginning to the end of the process.

As the rarefaction of the tissue goes on, the choroidal capillaries and smaller vessels of the choroid are absorbed, and the larger ones begin to make their appearance. The sclera begins to show through the thinned tissue, and little by little the absorbing process leaves a brilliant white figure of a crescentic shape, which embraces the outer edge of the nerve. In the early stages of the process the color of the crescent is not a pure white, but a yellowish or dirty white, which becomes clearer and more brilliant as the trouble progresses. The shape of the nerve is somewhat affected by the crescent, at least at the first glance, when the disk appears to be enlarged in a horizontal direction. Still closer examination shows that precisely the opposite is true, as regards the shape of the disk itself, independent of the added crescent, for under a closer examination the true boundary of the disk can always be determined, and it is then seen that, instead of being elongated horizontally, the nerve appears to be an oval, elongated even more than in the normal eye, in a vertical direction. This is due to the fact that, as the eye elongated, the outer edge of the nerve was drawn back, so that we see the nerve fore-shortened, just as a circle would appear oval, and an oval more oval, when rotated on its vertical axis. As a rule, the extremities of the crescent do not pass what would be the median line of the disk. Occasionally, however, this may occur, and the horns gradually extend round the entire circumference of the nerve until they may meet together on the opposite side; or there may be a second crescent of a smaller size, formed at the inner border of the nerve, and the two may then advance until their extremities meet at the upper border of the nerve, which then appears surrounded by an oval-shaped ring, with its long diameter

in a horizontal direction. Sometimes there are remissions in the advancement of the crescent, and then from some cause or other the process is renewed, and the tissue begins to become attenuated just outside the former place of atrophy, so that we have a fully formed and white crescent, bordered by a second one, in process of formation; and this may be repeated, so that the different accessions of the trouble may be recognized by a series of concentric markings, extending from the neighborhood of the nerve to that of the macula, not unlike the markings of a receding tide on the beach. As a rule, the crescent is situated directly outward. It may be, however, and not infrequently is situated obliquely, either upward and outward or downward and outward. Occasionally, but very rarely, it is found directly downward, and less frequently directly upward. I do not remember ever seeing it situated, when single, to the inner side of the nerve. This only happens, in my experience, when the second (inner crescent) is added to one already existing at the outer side. The extent of the crescent may vary from the merest trace to an enormous white plaque, which may extend far toward the macula. The original staphyloma rarely reaches the macula itself, because the macula moves synchronously with the cone, but it may be re-enforced by the changes that occur in this region, and which, coalescing with that which surrounds the nerve, the two foci coming together, may make an enormous oval, which extends even beyond the macula. Indeed, when complicated with what has been called sclero-choroiditis posterior, the staphyloma may extend in any direction. This white figure may then at times assume a very *bizarre* shape, and reach far out into the field like the wings of a butterfly, or assume a pointed or rectangular shape. In the early stages of the trouble the disk itself may show an abnormal injection instead of the pallor at the outer circumference just mentioned, and when this is at all marked the injection is, as a rule, more pronounced in the central portions of the nerve, and exhibits the characteristics that are common to choroidal rather than retinal irritation. The redness then appears to be at a more posterior plane in the disk, and the connective-tissue ring is even more marked than in the normal eye. In certain cases there is an excavation at the outer border of the nerve which may at times be of some considerable depth; when it is so it is usually accompanied by a shallower depression at its outer edge, or, indeed, there may be in some very rare cases a third, showing a shallower depression still. When such depressions exist, they usually vary in shade, the one at the disk assuming a bluish tinge, that next to it a pale white, and the most external, that toward the macula, a yellowish hue. Such

depressions strongly suggest a congenital defect, or an arrest of development, such as a coloboma and the imperfect closure of the scleral split with subsequent yielding, at a later date, to intra-ocular pressure or reduced resistance of the membranes. This would seem to be corroborated by the fact that Jaeger and Von Hasner have reported cases of marked and fully formed staphyloma in the eyes of infants, both in life and after death, and I myself have seen such with the ophthalmoscope. They are, however, very rare, while it can not be denied that the crescent increases in frequency in after-life, and with close application, and oftentimes in eyes which have been shown by repeated ophthalmoscopic examinations to have been free previously from the slightest trace of cone. From such facts, and from the fact that simple elongation of the globe would, from mechanical reasons, produce a crescent, it is very doubtful whether this is in any way the expression of congenital malformation of the scleral protuberance, or, indeed, of any part of the scleral split; for, if it had any connection with the split, we should suppose that it would be in reality what its name implies—that is, an actual protrusion of the sclera, or at least an excavation or limited thinning of this membrane, as is usually the case with a true coloboma, as demonstrated as well by the ophthalmoscope as by dissections. But with a true crescent there is never any difference in level between this part of the sclerotic and the adjoining portions, no matter how large the crescent may be. The cone, in the common acceptation of the term, is in fact not a true staphyloma, and it is only when applied to the entire posterior portion of the eye that the name is at all applicable; and we should be forced to assume that the whole posterior part of the eye was conjointly defective, or at least that parts were so that were widely separated from the scleral protuberance. There is another anatomical condition, which can hardly fail to have its effect in causing the elongation of the globe, and consequently the formation of a crescent. This is the peculiar continuation of the intervaginal sheath, which in highly developed myopic eyes splits the sclera for a considerable distance at the posterior pole, and especially at the outer side, into two layers (see Vol. I., p. 23). This split is simply a lymph space, and if under any circumstances, such as over-use of the eyes, the amount of fluid is increased within the space, or the resisting power of the sclera is diminished through a special or general debility, then the outer thin layer yields, and alternate overfilling and reduction of the space must stretch the membranes, and the inner layer then follows the outer. Thus, an elongation of the ball would take place, and with it progressive myopia and a progressive crescent;

with this elongation the outer sheath of the nerve would be drawn back, and that part of the sheath, which was in the line of the observer's vision, and thus not visible, would be stretched backward and become visible, and would then form part at least of the white surface of the cone. This would account for one of the concentric markings found at a little distance from the disk, within the surface of the crescent ("Stilling Zehender I. Monatsblätter," March, 1888). Thus we see that there may be two kinds of staphyloma—one, a true protrusion, which is exceedingly rare, and which is due to malformation, and which can be determined by the ophthalmoscope by the differences in the level of its floor, and by the bending of the vessels at the point of junction of the staphylomatous and non-staphylomatous portions; and one, which is vastly more frequent—which is, indeed, the typical staphyloma, cone, or crescent—and which is formed by simple extension of the membranes, in which there is no difference of level, and which produces in its turn a typical example of atrophy of the vessels through traction and pressure. As the crescent increases in extent, and particularly after it has reached the higher degrees of development, changes occur in other portions of the choroid; indeed, its entire surface may attain a faded and washed-out appearance due to changes and gradual absorption of the pigment, strongly suggestive of a senile wasting, and which is out of all proportion to the years of the subject. It is, however, at the macula lutea especially that these alterations occur; here the ame attenuation of the tissue, the same alterations of the pigment occur, so that the region of the macula has a mottled and abraded appearance, which finally may pass into an isolated spot of atrophy, which, strange to relate, may confine itself so strictly to the choroid that vision is not nearly so much affected as the ophthalmoscope would lead one to suppose. The pigment, however, is not so apt to collect in black masses as in the neighborhood of the nerve, but the whole region assumes rather a ruffled and macerated appearance. Occasionally, however, concentric masses of black pigment do occur, especially when associated with some inflammatory action. Under these conditions, also, hæmorrhage at the yellow spot not infrequently takes place, and we then have a very striking ophthalmoscopic picture, of which the drawing in the frontispiece of this work is so conspicuous and beautiful an example. This chromolithograph was kindly furnished to me by Dr. Hasket Derby, of Boston, and seems to me unsurpassed in the brilliancy of its execution and its fidelity to nature. It was taken from a case in Dr. Derby's practice, and represents the left eye of a middle-aged man, in which there was a myopia of 13 D. The patient had been near-sighted since his ear-

liest recollections. In addition to this, he had always been subject to marked intolerance to light. He had not, however, noticed any special failure of vision until a few weeks before he presented himself for examination, when a large central scotoma had appeared before this eye. Vision was eccentric in this eye, and reduced to ·03. In the right eye there was a myopia of 13 D., with a large and progressing crescent, with numerous isolated spots of atrophy scattered over the fundus, but there was no special change at the macula. In the left eye (see frontispiece, Vol. I.) there was a large crescent surrounding the nerve. There were, however, no isolated patches of atrophy in any other portion of the fundus, except the macula, where the appearance existed that is figured in the drawing, which was made by the inverted method. The effusion of blood is evidently quite recent. This patient was examined a second time, three years later, and the only ophthalmoscopic change had been a slight clearing up of the pigmented spot in the centre. It is evident, from the existence and persistence of the central scotoma, that the outer layers of the retina were affected in this case, as they are in so many like it; and it is more than probable that the hæmorrhage in these cases is from the retina, rather than from the choroid. In this particular case it is shown to be so from the interruptions in the vessels. This, then, is a case of chorio-retinitis centralis, grafted upon a posterior staphyloma, which would appear to be originally of congenital origin. The occupation of the man was that of switch-tender, and Dr. Derby justly remarks that "this case furnishes an instructive commentary on the necessity of submitting all railway employés to an examination of vision. For years before coming to me he had occupied a responsible position on one of our most important roads, and had been daily intrusted with the carrying out of arrangements to which accurate eyesight would seem essential, and which involved the safety of hundreds of travelers." Just as posterior staphyloma may be anatomically divided into two classes, in one of which there is a real protrusion of the sclera, due to defective formation, and in the other a simple ellipsoidal distention of the posterior portion of the eyeball, so clinically it may be divided into two classes, the stationary and progressive. The stationary occurs either congenitally or very early in life, increases with the growth of the body, and then becomes stationary, and may remain so for life, with little or no apparent ophthalmoscopic change other than that which is common to all eyes with advancing years. These stationary staphyloma may be of considerable extent even from birth. Such eyes are, however, from the very nature of things, apt to take on a sudden development, and pass from being stationary into progressive.

Stationary staphyloma usually occurs in those people who have been myopic from their earliest recollections, and from those of their parents, who are themselves apt to be myopic. These stationary staphyloma or crescents, have usually ophthalmoscopic peculiarities which distinguish them from those of a progressive nature. Thus, the border of the crescent, where it meets the choroid, is sharply defined, and there is usually little or no disturbance of the pigment, and the adjacent choroid shows little or no signs of attenuation or beginning atrophy, the tissue appearing normal, quite up to the divid ing line between it and the staphyloma. This is well seen in the frontispiece to Vol. I., where there is a perfectly clean-cut edge to the staphyloma, with no trace of pigment or rarefaction of the tissues. That a secondary process, of an inflammatory character, was superadded to the staphyloma, which also affected the retina, has already been explained. Moreover, the staphyloma is not usually of so regular and typical a form—that is to say, of so crescentic a shape. Again, its position is not apt to be so predominating or dense at the outer edge of the nerve, but may not infrequently assume a less common locality, such as downward or upward, or obliquely outward, or even inward. These stationary staphyloma are apt, moreover, as many if not most of them are congenital, to have a shallow excavation at the outer part of the nerve, which passes over into the staphyloma; or there may be a second, or even a third, shallow difference in level, with a different shade of color in each. In a word, the crescent has rather the appearance of a long-standing trouble of a congenital character, and is strongly suggestive of coloboma, than the result of an atrophy produced by an existing pressure and traction. As elongation of the eyeball produces myopia, and also produces the crescent, the terms progressive myopia and staphyloma have become almost synonymous. This is no doubt true, in the main, although it must not be lost sight of that myopic eyes occur, not infrequently, where there is no cone, even where the myopia is of a considerable degree. This occasionally happens, even where the myopia is of a high degree; thus, I have not infrequently seen myopic eyes, without a cone, where the myopia equaled 9 D., and a few even where it was as high as 12 D. Neither must it be forgotten, on the other hand, that the cone may occur in eyes which are not myopic; thus, it is found, in about three per cent of emmetropic eyes, and in about three and a half per cent of hypermetropic. In such eyes the crescent is usually of small dimensions, is generally also stationary, and may, in the great majority of cases, be looked upon as congenital.

As to the cause of the formation of the crescent, it may be said,

in general terms, that it will result from any cause that produces an elongation of the eyeball, and which at the same time produces sufficient pressure and traction upon the vessels of the choroid to cause their atrophy. That the resistance to such atrophy varies in different eyes is shown by the fact that in some cases of progressive myopia, especially in youth, considerable elongation can take place without the production of any crescent. That heredity plays an important part in the production of myopia and the crescent can not be denied; but that they occur in eyes where there is no hereditary tendency is equally true, and where its origin is purely fortuitous, as in over-use of the eyes, after debilitating diseases. It is often associated with a low form of choroiditis which is located in the posterior parts of the eye, and there has gained the title of "sclero-choroiditis posterior." In this case it may be associated with patches of choroiditis disseminata, either isolated or confluent; or the process may be of so low and chronic a type as to show few or no signs of active inflammation, either to the ophthalmoscope or microsope, and be characterized solely from the atrophic variety by the enormous extent over which the degeneration and wasting away of the tissues occur. In the most marked cases of this, one or more foci of atrophy occur, that are situated especially at the optic nerve and at the macula, plainly showing that the vessels of the scleral circle, and those which pierce the sclera in the neighborhood of the macula, are affected. In this way two large circles are formed, which coalesce finally and form an immense oval or figure-of-eight patch, which embraces the entire circumference of the nerve and the macula. (See Fig. 62, which represents a case of marked sclero-choroiditis posterior.) The appearances represented were found in the left eye of a young girl who was suffering from progressive myopia, the result of overwork at school. The patient was myopic, 9 D, and the fundus showed the signs of a well-marked posterior staphyloma of that rather rare form which surrounds the entire nerve; and the line of separation between the nerve and adjoining sclera was so faintly marked as to give the impression of an enormously large disk, upon which the ramifying vessels stood out with great clearness. I have, indeed, as a rarity, seen cases in which the degeneration seemed to cover almost the entire fundus, and which might be fairly looked upon as cases of complete atrophy of the choroid, the fundus being marked here and there with streaks and circles of black pigment, and occasional bits of attenuated vascular areas, which sometimes have the appearance, when very small, of hæmorrhages, but which on closer inspection are found to be made up of small vessels which have escaped the general eroding in-

fluences. Real hæmorrhages, however, do sometimes occur, minute in form, and situated near the border of the staphyloma. These are rapidly absorbed, as a rule, showing that they were really extrava-

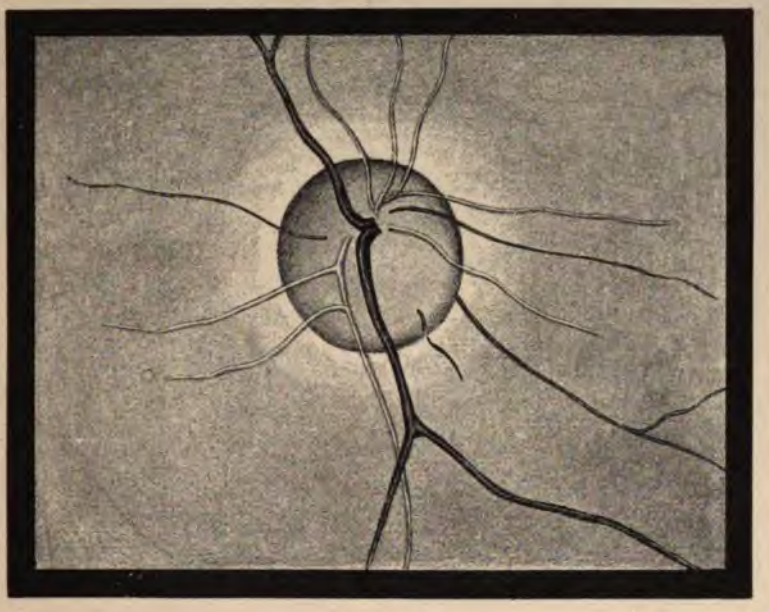

FIG. 62.

sations. The retina is not nearly so much affected, in elongation of the globe, as would naturally be suspected, from what is usually considered its extremely delicate nature. In the earlier stages and in the milder forms there is often, indeed as the rule, no change that can be detected with the ophthalmoscope, and the function of the retina may be absolutely unimpaired, vision being normal in every respect. In the higher degrees the most conspicuous change is, that the vessels seem to run a straighter course and to be smaller than in the normal eye. Some of this is due to the fact that the nerve entrance, together with the staphyloma, is so much larger, that in comparison they seem reduced in size; but, independent of this, it does seem in other parts of the fundus, actually as if, in being stretched, they had lost some of their calibre. In the higher degrees only, vision is decreased, as a rule. In the more marked forms, where the myopia is of a considerable degree, the vitreous is apt to be the seat of long filiform membranes, which are sometimes very distressing to the patient. When situated in the back of the eye, and not in the anterior portion, and when they make large and rapid

excursions on movement of the eye, they are thought to indicate a greater liquefaction of this portion of the vitreous. Occasionally also there is what appears to be a posterior polar cataract in the lens. This usually takes the form of a small dark spot on the apex or curvature of the posterior capsule of the lens. When, as often happens, this opacity is exactly covered by the corneal reflex, and maintains this position under all movements of the eye, there is every probability that it is not in the lens itself, or its capsule, but that it coincides with the centre of motion of the eye which is behind the lens and in the vitreous body. It is well, then, to determine its position acurately, and not to mention the existence or probable formation of cataract, as I have known such spots to maintain their size and position without alteration for years (see Vol. I., p. 187). That such eyes should show a tendency to mal-nutrition in all respects is not strange, nor that they should be subject, as they often are, to cataractous formation and separations of the retina and other degenerative changes.

Microscopical Changes.—The chief of these are degeneration or atrophy of the pigment, atrophy of the chorio-capillaries, and other vessels, and a gradual reduction of the choroid, which makes it, in the position of the posterior staphyloma a thin membranous expansion, some traces of which always remain in the epithelial layer and choroid proper; there is also cohesion of the retina to the choroid, and the choroid to the sclera. There is also in some forms sclerosis of the vessels.

Changes in the Epithelial Layer.—Midway between the choroid and retina stands the remarkable anatomical structure known as the epithelial or pigmental layer. Embryologically it would appear to belong to the retina, and clinically to the choroid. Always remarkable as an important factor in the ophthalmoscopic picture, it has gained new importance and significance from the fact that it secretes the visual purple, and may therefore, as Alt has expressed it, be looked upon in the nature of a gland. Situated immediately between, and in close contact with the retina on one side and the choroid on the other, it would only be natural that it should participate in the morbid changes to which both of these principal membranes of the eye are subject. On the other hand, this thin and apparently delicate membrane would appear to form a protecting lamina for the prevention of the spread of disease upon either side of it: thus we often see the most marked forms of choroiditis which do not implicate the retina; and, on the other hand, cases of retinitis in which the choroid remains intact. The epithelial layer may be considered, so far, as

divided into two parts, one of which consists of a thin vitreous layer, and the other of a single layer of pigment cells, which are spread over its inner surface in the manner already described in Vol. I., p. 37. Each of these may undergo changes visible with the ophthalmoscope. Thus, there is no doubt but that in certain forms of choroiditis and retinitis, especially in the outer layers, the pigment cells may proliferate and be altered in shape, and that certain cells may lose either a part or the whole of their pigment, while neighboring cells may become so engorged as to be completely filled with coloring matter. As will be seen later with the choroid, as has already been seen with the retina, these changes are a constant accompaniment of certain forms of choroiditis; whether, however, there may be morbid processes which affect the pigment in an independent and idiopathic manner, without some complication of either the retina or the choroid, is not yet settled. Yet it can not be denied that there are certain collections of pigment which take place, especially at the macula lutea, in which there are no traces of any action which can be classed as inflammatory, to be detected at the time, or for years afterward. As such accumulations of pigment are usually accompanied by a deterioration of vision, in the shape of a scotoma, they have been usually set down to some preceding hæmorrhage. Single hæmorrhages into the retina, as a rule, do not produce these deposits of pigment, and there is probably, as Alt says, a hyperplasia of the pigmented epithelium without a pathological change in the choroid; and that in such cases the tubular offsets of the pigmented epithelium grew into the retina after having first destroyed its outer layers ("Lectures on the Human Eye," p. 123).

This hyperplasia of the pigment, together with its displacement in certain places and its agglomeration in others, would form an image, that would resemble, in a certain respect, choroiditis disseminata; only the denuded spots would not be so conspicuous. On the other hand, in certain cases, it would take on the appearance of retinitis pigmentosa, though in these cases, as the conducting elements are not apt to be affected, there is, as a rule, but little or no defect in vision or field. This form of inflammation usually begins in the periphery and progresses toward the posterior pole. In very early life we sometimes see a peculiar disturbance in the pigment layer, that consists of exceedingly minute spots, which look precisely as if the fundus had been peppered over with a very fine and intensely black pepper. Such eyes have usually an intolerance of light, with no perceptible or only hardly perceptible changes in the choroid or retina. Occasionally, however, the general fundus has a faded or

washed-out aspect, which gives a "pepper-and-salt" character to it. Whether these conditions are a preliminary step to some ulterior and grave process, I have never been able to determine. This appearance sometimes, but not universally, is found in the fundus of grown people. In some asthenopic eyes, even in those where there is no error of refraction, there is a certain macerated appearance of the pigment, as if minute particles of it had been dislodged from their resting-place and carried along with a current and deposited where they did not belong. Thus, we often see in the anterior portion of such eyes long strips of a more rarefied tissue, with the appearance as if the coloring matter had "run," as we sometimes see it in a water-color. Also, at times, we find a granulated effect, as if the epithelial surface had been roughened, and which was due, not to any inequalities in the vitreous lamina, but to a displacement and alteration of the cells of the layer itself, as if, indeed, there had been a hyperplasia of cells, partly of those bearing pigment, and partly of those which did not contain coloring matter, and which gave a sort of roughened appearance to the fundus, like certain kinds of coarse cloth. This happens particularly in the region of the macula, where a little cluster of these formations may exist, which have the appearance of abrasions of the surface. Such a condition is not usually accompanied by any loss of vision. Besides these unformed and indistinct granular configurations there are sometimes very minute spots of a pure yellow color, not larger than a pin-point, of which it is hard to determine whether they are in the retina or choroid. These seem to have no effect upon the vision or the functions of the eye. They strongly suggest the fatty degeneration which, according to Wedl, may also occur in the pigment cells of the epithelial layer, but their true nature is not yet determined, at least to my knowledge. In serous choroiditis this striped and roughened aspect of the choroid is well marked, when the media remain clear enough to permit their examination.

Atrophy of the Pigment Layer.—If there may be an independent hyperplasia of the cells of the pigment layer, there is no reason why there should not be an idiopathic atrophy as well, which might extend over large districts, or the entire membrane, and might also contribute to that faded and washed-out appearance which the choroid presents as well in some diseases, as the result of senile degeneration. I know of no way, however, of determining such a condition with certainty. Jaeger originally applied the term "atrophy of the pigment layer" to a very conspicuous ophthalmoscopic picture in which, through the atrophy, as alleged, of the epithelial layer, one was enabled to see plainly the stroma of the choroid, and the curves and anastomoses of

the choroidal vessels. Fig. 63 (Jaeger, "Hand Atlas," Taf. XXII., Fig. 97) represents such a condition, but it is very evident that the lesion is much more complex than a simple atrophy of the pigment epithelium, and that it comprises also atrophy of the chorio-capillaries and the small vessels, and alterations of the walls of the large vessels, probably due to sclerosis. The outer border of the atrophic patch is clearly defined, while above, and to the outer side, the inner border shows a delicate and transparent veil-like extension, as if the atrophic process had not advanced to so great a degree as in other parts, but sufficient to allow the larger vessels to be seen through it. That the retina is also implicated is shown by the fact that there was a central scotoma and a considerable reduction in vision, as well as the fact that the connective-tissue ring is obliterated. This condition, then, really is one of circumscribed and non-exudative retino-choroiditis, in which the outer layers of the retina, the epithelial layer, and the finer vessels of the choroid are more or less atrophied.

FIG. 63.

Perivasculitis and Sclerosis of the Choroidal Vessels.—The condition of atrophy just described permits us to see at times a complication of the trouble which, in some cases, would appear to be due to perivasculitis, and in others to a sclerosis. In the case of perivasculitis the walls are much thickened, as in a case reported by Dr. Knapp, in which there were floating opacities in the vitreous, staphyloma posterior, and superficial atrophy of the choroid. Here the scarcity of pigment in the eye admitted of a thorough examination of the choroidal vessels, even where there was no atrophy. Some of the larger vessels had sharply cut white margins, which were broader than what we see in cases of marked perivasculitis ("Trans. American Ophth. Soc.," 1869, p. 120). Nettleship has also reported a case under the designation of "Central senile areolar choroidal atrophy," in which perivasculitis of the vessels would appear to be well marked.

The patient was a man of sixty years old, whose sight had been failing for twelve years, but which had only decreased, in a marked manner, for the past four months. In each eye there was a large area of atrophied choroid, which occupied the central region of the fundus. The drawing (Fig. 64; see also chromo-lithograph, Plate VII, Fig. 1) is from the erect image of the left eye, and shows the large extent and completeness of the atrophy. The conspicuous thickening of the coats of the larger vessels is plainly seen, and it will be noticed that, while the borders of the vessels are lined with white tape-like strips,

FIG. 64.

the centre maintains its normal red color. The fine white vascular branches are vessels which have probably been completely obliterated ("Trans. Ophthal. Soc., United Kingdom," Vol. IV., p. 165). In sclerosis of the vessels in contradistinction to perivasculitis, the entire diameter of the vessel has a yellowish-white appearance. The borders of the vessels are not so sharply limited by white bands, and the vessels themselves are not increased so much in diameter, if at all, and look like ordinary vessels, but of a lighter color.

The Vitreous Lamina.—Under certain conditions the inner surface of the lamina vitrea becomes the seat of minute elevations, which

are usually known as "colloid bodies," though their exact chemical constitution is not yet known. These excrescences are much more frequent in advanced life, as a senile manifestation, than in youth. These minute elevations are usually found more frequently at the equatorial regions of the fundus than elsewhere, and are not infrequently, however, situated at the region of the macula, and occasionally at the circumference of the nerve. Although these are the places of predilection, they may be found in other parts of the fundus, especially along and beneath the course of the larger vessels after some forms of retinitis. They may vary much in size, from a small light-colored body, which can hardly be recognized with the mirror, to a well-formed projection as large as a millet-seed. They are usually of a light-yellow shade, and of a roundish shape, and are so closely studded together as to give a tesselated appearance to the portion of the fundus over which they extend. As a rule, the pigment layer does not seem to be much disturbed, and the coloring matter is not collected to a perceptible degree in the interstices of the neighboring bodies. It gives the general appearance as if, in growing, they had gently displaced the pigment layer to one side, or that the pigment layer immediately over their summits had been absorbed by pressure, while that lying between them had not proliferated to a considerable degree, though occasionally their boundaries are accentuated by an increased degree of coloring matter. With the ophthalmoscope, in old people the fundus has a mottled appearance, and shows a play of light and shade under movements of the mirror, as if the surface was slightly indistinct or roughened. These inequalities may occur after some forms of retinitis, especially those which attack the outer layers, and they are evidently the secondary result of the retinal disease, as they occur sometimes at the very end of the trouble, or even after all traces of the primary lesion have disappeared. The fundus may then at times appear as if studded with fine drops or semi-transparent vesicles. In some forms of retinitis, especially when due to syphilis, these colloid bodies attain a considerable size, even in young subjects, and are then grouped immediately round the border of the optic nerve, at the posterior pole, and at other regions. Under the title of "Syphilitic choroido-retinitis, with peculiar growths at the fundus," Mr. Lawson has given a remarkable case, which would appear to be of colloid origin. The peculiar and bead-like formation not only surrounded a portion of the circumference of the nerve, but also at a little distance from the disk penetrated the retina and projected like a cluster of grapes into the vitreous ("Trans. Ophthal. Soc., United Kingdom," Vol. III., p. 117). Microscopically, these ex-

crescences appear to consist of transparent elevations, which have a concentric construction, surrounded by a line more or less marked with pigment. They sometimes project to a considerable degree into the retina, and remain covered with numerous pigment cells. As is shown by Alt, they may contain calcareous masses, and even true bony tissue, and thus, according to the same author, these vitreous bodies may be the starting-point of osseous plates upon the inner surface of the choroid ("Lectures on the Human Eye," p. 135). It has not yet been determined what their true nature is. The supposition that they are really of colloid substance would not appear to be borne out by either their consistency or their reaction to chemical reagents.

Irritation of the Choroid.—I am of the opinion that irritation of the choroid is a condition which may and does not infrequently occur in eyes which suffer from overwork or exposure, and which may justly fall into the broad and, it must be admitted, very indefinite term of asthenopia, especially in those forms of asthenopia not dependent on errors of refraction or muscular insufficiency, although it may and frequently does coexist with them. The principal and sometimes the only ophthalmoscopic sign then manifested is situated not in the membrane itself, but at the disk. This irritation then manifests itself in a peculiar congestion of the surface of the disk, which is more apparent in the central portions of the papilla, in the region of the connective-tissue string which surrounds the central vessels. This congestion is in a plane posterior to the surface of the retina and in a plane with that of the choroid proper, and is marked by a peculiarity which is distinctive of choroidal congestion, namely, that the connective-tissue ring at the outer border of the nerve is not obscured as in retinal irritations, but is, as a rule, more conspicuous. The choroid proper is not usually affected, to a degree at least which manifests itself to the mirror. It usually preserves its normal appearance, and maintains that firm tone in contradistinction to that soft and hazy atmosphere which it takes on in a more advanced state, and which may be designated as hyperæmia of the choroid. Exceptionally, however, the pigment layer may be implicated, and show that washed-out and faded and streaky appearance, especially in the outer portions of the field, which has been already alluded to in discussing the pigment epithelium.

Hyperæmia of the Choroid.—This may be looked upon as an advanced stage of irritation, in which there is actual overfilling or hyperæmia of the vessels of the stroma of the choroid. While admitting that hyperæmia of the choroid must exist in many forms of eye-

disease, and that this may and often does manifest itself by that peculiar congestion of the disk and increased conspicuousness of the connective-tissue ring, most observers deny the ability to detect any evidence of hyperæmia of the choroidal vessels in the membrane itself, though this has been done.

Hæmorrhage into the Choroid.—It is certainly very curious that hæmorrhage into the choroidal tissue is not often seen with the ophthalmoscope. It will be said at once that the reason is, that the epithelial layer prevents this, and that this barrier to further penetration of our view is also aided by the denseness of the vascular supply and the nature of the stroma. Admitting that this is true in some cases, there are surely enough eyes, where the epithelial layer is wanting and where the vessels are plainly seen, as in blondes, especially at the periphery of the field, in which extravasations ought to be plainly visible, especially as we not infrequently get a reflex even of the sclera. That such extravasations are visible at times can not be doubted, but at the same time they do not occur as frequently as one would suppose in a membrane which is made up of vessels, or as they do in the retina, which is a tissue poor in blood-vessels. There must be something in the construction of the walls that prevents an easy rupture and an easy diapedesis. When the hæmorrhages occur, they are said by some authorities to be of a rounder shape than those which take place in the retina. This, however, is by no means always the case, as I have seen radiating extravasations even in the choroid-notably in one case—the result of a heavy blow and rupture of the anterior portion of the sclera. Strange to say, the media remained perfectly clear, and I could follow a rather profuse hæmorrhage in the choroid from the anterior part of the field along the inner wall of the eye. This was linear in shape, and extended posteriorly nearly to the disk. It had a somewhat subdued aspect, but could be differentiated plainly by its color from the rest of the fundus, and was evidently in the outer layer of the choroid. The diagnosis as to the hæmorrhage being in the choroid, and not in the retina, is of course helped by the fact that the retinal vessels would pass over it, and can be seen to lie in a plane anterior to that of the extravasation; and especially is this confirmed by the fact that paralactic displacement between the vessel and the hæmorrhage can be obtained. With all cases it is sometimes very difficult, if not impossible, to tell whether a hæmorrhage is in the inner layers of the choroid or the outer layers of the retina, or in both. Should this occur in the posterior pole of the eye, a diagnosis may be inferred at least, by the determination of the vision, and especially by the presence of a scotoma when the

hæmorrhage is in the retina, and the absence of these when it is in the choroid.

Serous Choroiditis.—Serous inflammations of the uveal tract, such as serous iritis and serous choroiditis, are usually accompanied by opacities on the posterior surface of the cornea, and in the media themselves, to such a degree that the fundus is usually hidden from view. This is particularly so with serous choroiditis, so that the ophthalmoscopic signs in the early stages of the disease are rarely—it may be said never—seen, except in the very moderate forms, when the appearances are simply those which have been described as sometimes occurring under hyperæmia of the choroid. When, however, the acute stages have passed, and the media become clear enough to permit an examination, the fundus is found to have undergone extensive changes of an ill-defined but perfectly recognizable character. There are no well-defined spots of exudation or atrophy, surrounded by rings of blackened pigment, as occurs in parenchymatous choroiditis; but the choroid presents a macerated appearance, with a rarefaction of the tissue, which is very expressive of the disease, with large areas of faded, washed-out tissue, upon which are small dots and long streaks of dust-like accretions of pigment. This takes place especially in the anterior portions of the eye, and is then probably due to propagation from the iris and ciliary region. It may also occur, however, at the posterior pole, or indeed in any part of the fundus. It is found most frequently with specific troubles, and then may be accompanied by a low form of retinitis, in which the borders of the nerve are obscured. There is also apt to be a light mist-like opacity in the posterior part of the vitreous, with dust-like aggregations, and the trouble under this mixed condition may be fairly called a chorio-retinitis serosa. Vision is apt, in this form of disease, to be much affected; it is also apt to recover and relapse frequently, rather suddenly passing from a fair state of vision to a very high degree of amblyopia. Serous choroiditis may lead to separation of the retina detachment of the vitreous and glaucoma, and to it may be attributed that faded and irritated air that the fundus so often wears in chronic glaucoma, or as secondary results after acute attacks.

Microscopically, according to Alt, the serous exudation is mostly found inward from the choroid, between the lamina vitrea and the pigmented epithelium, or between this latter and the retina, or between this again and the vitreous. There may be, also, small round, or oval cavities in the outer granular layer of the retina, filled with a sero-fibrinous fluid, due directly to disease of the choroid before a secondary affection of the retina can be developed ("Lectures on the

Human Eye," p. 126). The position of the fluid in the innermost layer of the choroid and epithelial layer, would account for the soft and out-of-focus look that the fundus has in hyperæmia and serous troubles of the uveal tract.

Plastic or Fibrinous Choroiditis.—This comprises all the various forms of choroiditis which, from clinical appearances, have been divided into choroiditis disseminata or exudata, areolaris centralis, and atrophic; and, when associated with retinitis, they are further designated as chorio-retinitis disseminata, etc. They all belong, however, to one class anatomically.

* * * * * * * *

NOTE.—This finishes all the material left by Dr. Loring, with the exception of a few notes on Glioma of the retina, which were too blurred to be deciphered and too fragmentary in their nature to be available. The reader may have also noticed that in the earlier parts of the book the refraction has been expressed in the inch system, while in the latter the metric has been used. This is due to the fact that it was written at different times, when the two systems were in vogue; and I have let them both remain, first, because the student can convert them for himself at a glance, and, second, because what is true in large things holds good in small, and it has been my one aim and object to present this work, as far as possible, exactly as the author left it.—EDITOR.

PLATE I.

Fig. 1. *Phlebitis.*—The attack came on suddenly, after vomiting, from an overdose of quinine. There was no pain, or other subjective symptom; slight blur in upper field of vision, V = 1. There was a quadrant of field scotoma, which was thin and gauze-like, but the actual loss of vision was only at the periphery of the field where the dark shading is. (See text, page 7.)

Fig. 2. *Anastomosis of Vessels.*—This presents not only an anastomosis of the vessels, but what would appear to be a localized collateral circulation. It occurred in a young man who was suffering from retinitis syphilitica. There is an extensive hæmorrhage, which nearly covers the entire disk. In the upper and outer field there is a large yellowish-white plaque, over which runs a mesh of vessels, which evidently communicate with each other. There is no elevation of the surface, and the plaque is in all probability the remains of a blood-clot which was in the process of vascularization. (See text, page 39.)

PLATE I

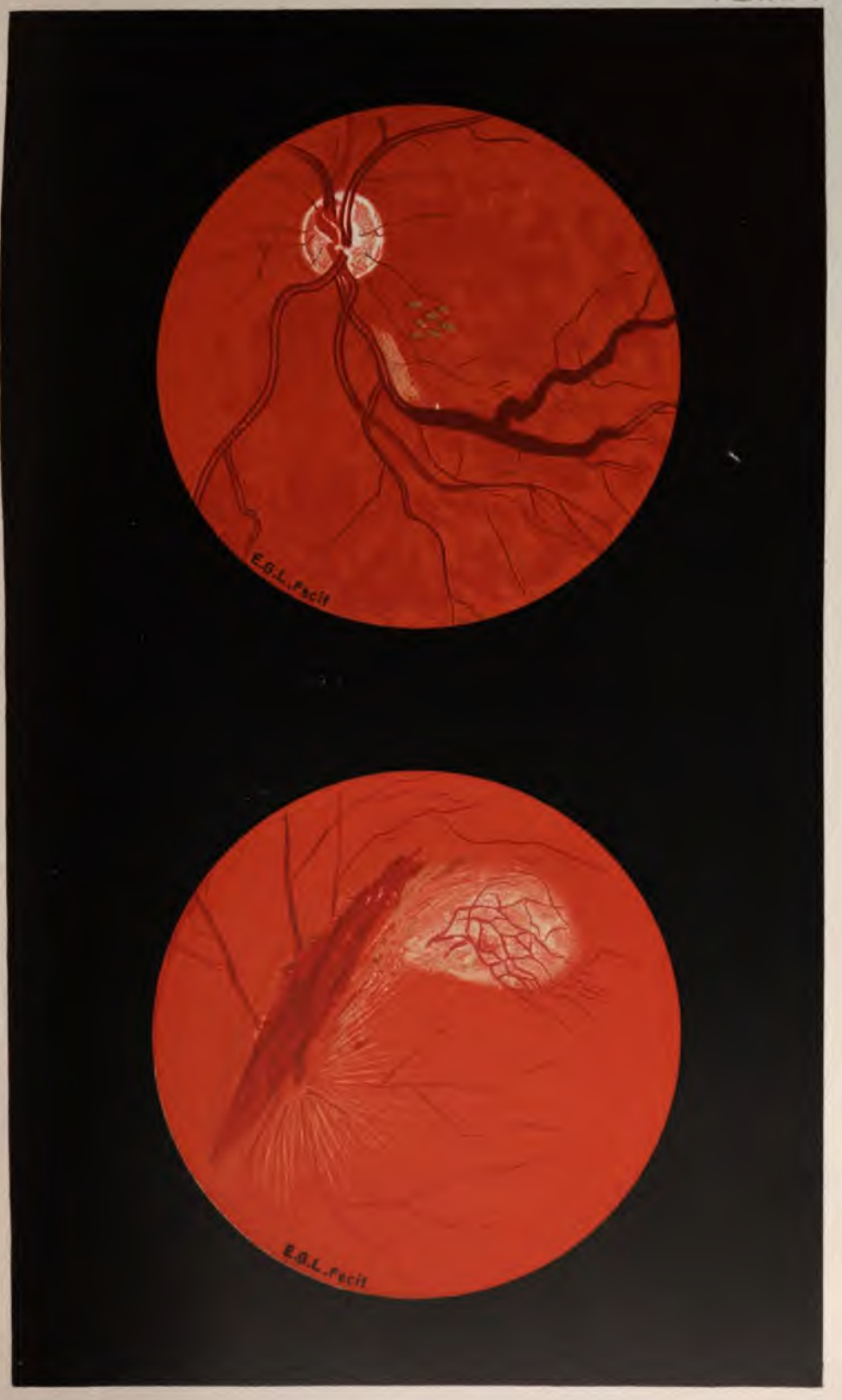

PLATE II

PLATE III.

Fig. 1. *Embolism.*—This figure represents the same eye, some six weeks after the attack. As will be seen, the vessels have gradually diminished until the secondary branches have in some places faded away, or have become of thread-like thinness, while along the principal branches longitudinal white bands are formed, and there is a hyperplasia of connective tissue in and around the nerve, which together with the retina is passing into a state of atrophy. In two places a thread-like vessel can be followed from one of the larger branches of the upper and lower veins for a considerable space, when it very rapidly increases in size for a short distance, to become again suddenly filiform and then untraceable. As the rest of the vessels are either empty or nearly so, these distended portions are in all probability blood-coagula within the walls of the vessels. (See text, page 82.)

Fig. 2. *Embolism.*—In this case the retinal vessels, neither veins nor arteries, show any marked deviation, either in size or course from the normal, except that the "light-streak" is reduced in brilliancy, and near the disk is entirely absent. From the outer edge of the disk, however, straight out to and including the macula, there runs a grayish-white patch, which has the appearance of a dense exudation into the retina. The position of the yellow spot is marked by a perfectly circular bright-red spot. Through the centre of the effusion, from the edge of the disk to the red spot at the macula, runs a small vessel, which is probably simply an increase in size of a pre-existing vessel. (See text, page 94.)

PLATE III

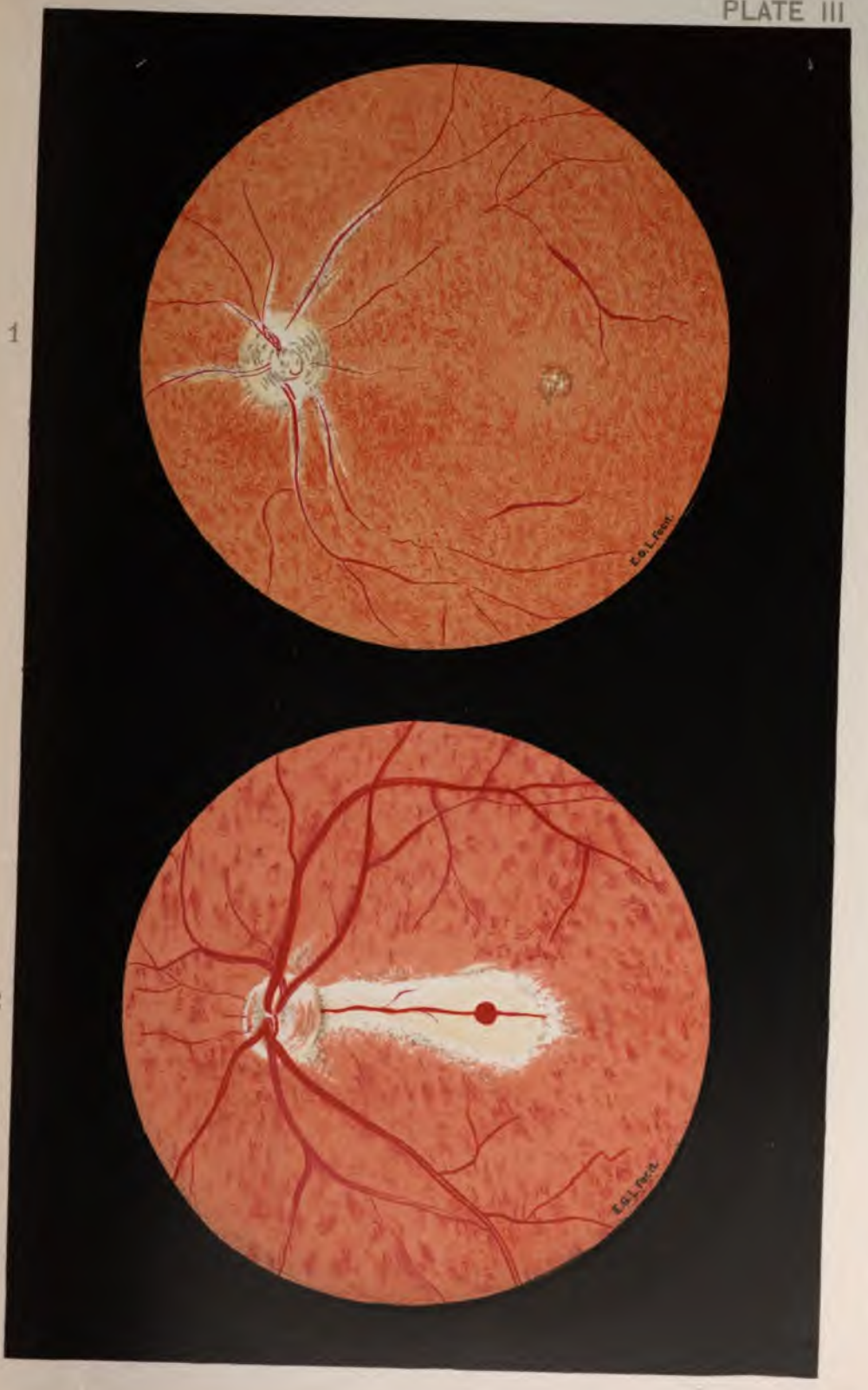

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PLATE IV.

Fig. 1. This figure represents the same case a month after the attack; the exudation has been gradually absorbed, while, in the region of the macula, spots, which appear to be due to fatty degeneration, begin to make their appearance; and the vessels, from having their normal calibre, gradually fall away in size, while longitudinal white bands begin to develop themselves along their larger branches, and a general atrophic condition has taken place. (See text, page 96.)

Fig. 2. *Retinitis Albuminurica.*—This figure shows a case that has passed from a state of simple retinitis to that of a neuro-retinitis. About the papilla are large yellow masses arranged in a circular form, but at a little distance from it. (See text, page 147.)

PLATE IV

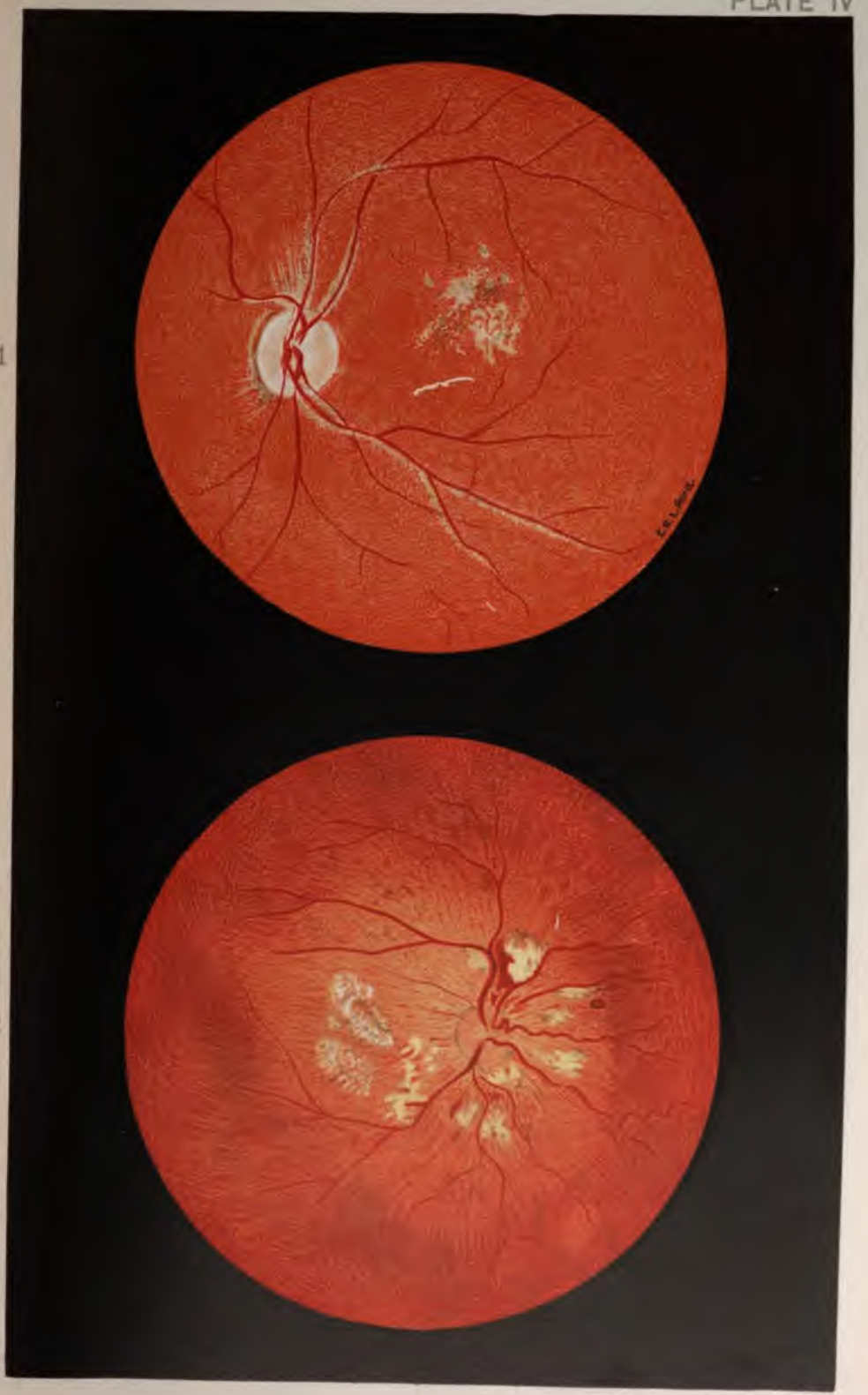

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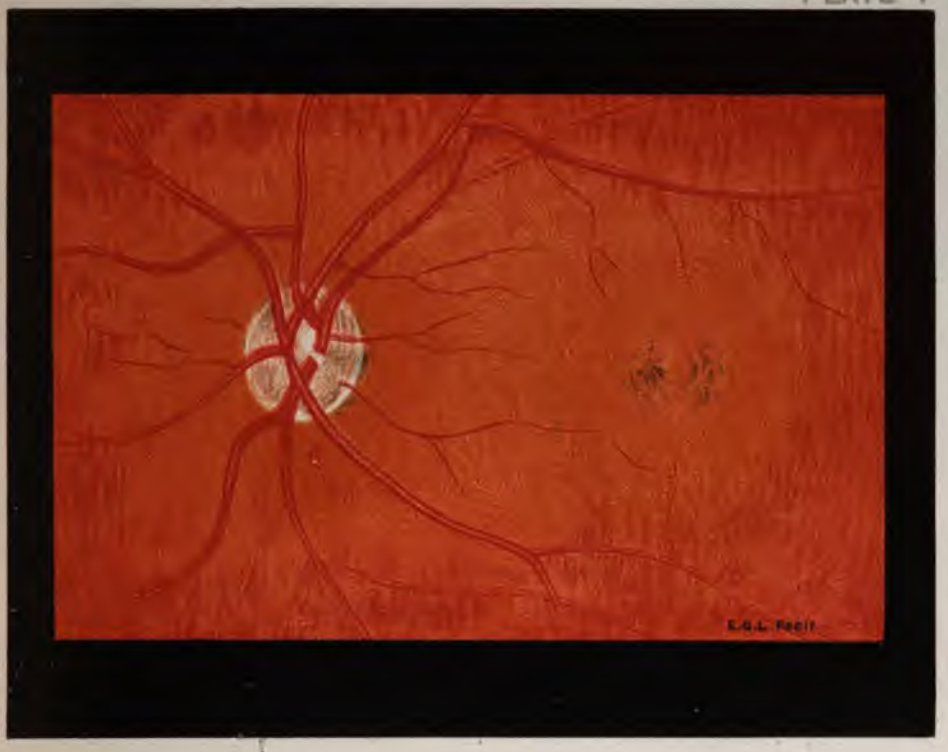
PLATE V.

Fig. 1 represents a myopic eye, in which there has been an attack of that form of retinitis, at the macula, which seems not only to be in the outer layers of the retina, but also in the pigment layer itself—a sort of hypertrophy of the pigment cell, which shows itself by a collection of pigment at and around the fovea, accompanied by few or no other inflammatory symptoms. (See text, page 139.)

Fig. 2. This figure shows a case of retinitis, in which the small central spot at the fovea was surrounded by a circle of still more minute spots, which gave a glittering reflex, as if they were due to some lustrous substance. (See text, page 140.)

PLATE V

1



2

PLATE VI.

Fig. 1. *Retinitis Punctata Albescens.*—Taken from a young woman of twenty-five years. This shows the characteristic spots, and also the absence of any other active inflammation. (See text, page 142.)

Fig. 2. *Syphilitic Retinitis.*—This represents a case of syphilitic retinitis occurring in a young man of thirty years. Through the atrophy of the epithelial layer the fundus has acquired the appearances of a diffuse choroiditis, with marked changes in the pigment and stroma. The sketch at the bottom shows the development of flecks of pigment, which differ from those of retinitis pigmentosa, as they do not have the characteristic bone-corpuscle shape. (See text, page 145.)

PLATE VI

1

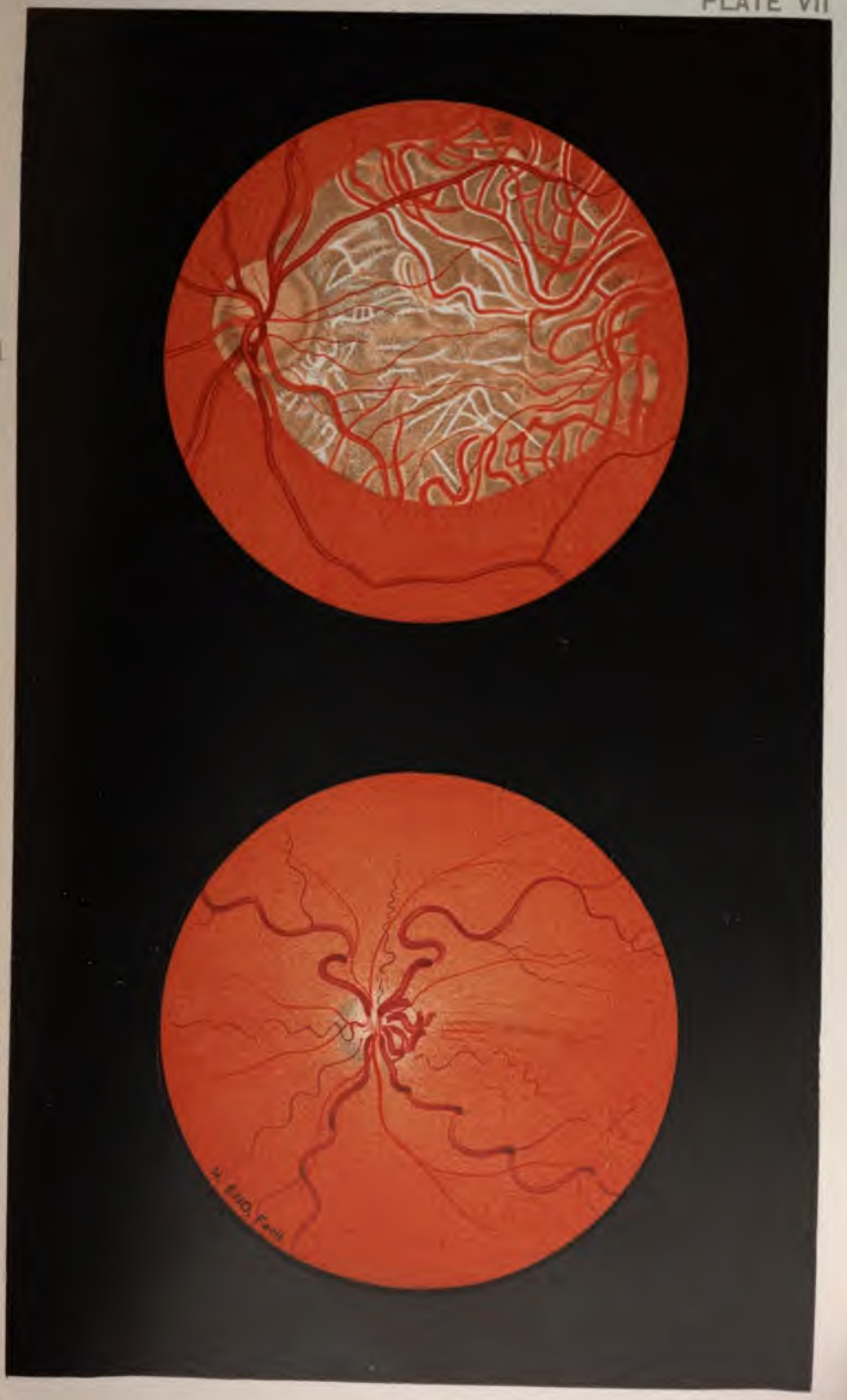
2

PLATE VII.

Fig. 1. *Central Senile Areolar Choroidal Atrophy.*—This is from a case reported by Nettleship, of a man sixty years old, whose sight had been failing for twelve years, but which had only decreased in a marked manner for the past four months. In each eye there was a large area of atrophied choroid, which occupied the central region of the fundus. The drawing is from the erect image of the left eye. The disk is situated on the nasal border of the diseased area. Over the nasal portion of the area the atrophy is complete, only a few white lines seen against the slightly gray background remaining to indicate obliterated blood-vessels. Toward the temporal side the atrophy becomes gradually less marked, and on this part very conspicuous thickening of the coats of the large vessels is noticed—a change which no doubt passes on to their complete obliteration. The diseased area is sharply defined everywhere, except at the extreme outer part. The choroid bounding its lower border is darkened, possibly by capillary congestion, but with this exception there is no evidence of inflammation preceding the atrophy; nor is there any accumulation of pigment. The first change seems to be the disappearance of the chorio-capillaris; this is well seen at the right-hand lower part of the drawing, where, although the large vessels show as yet but little change, the limit of the disease is marked by the sudden cessation of the "stippled" appearance produced by the epithelial and capillary layers. The disk shows no marked change, and the retinal vessels are normal. (See text, page 248.)

Fig. 2. *Choked Disk.*—This is a drawing by Dr. Eno, and represents the fundus of an eye in a case of choked disk from tumor of the brain. The tortuosity of the vessels is well shown, and also the protuberance of the head of the nerve—the refraction at the disk, as measured by the ophthalmoscope, being + 3.5 D. (See text, page 195.)

PLATE VII



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